THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—20TH YEAR. SYDNEY, SATURDAY, JUNE 24, 1933. No. 25.

Table of Contents

[The Whole of the Literary Matter in THE MEDICAL JOURNAL OF AUSTRALIA is Copyright.]

ORIGINAL ARTICLES— PAGE.

An Address, by FRANK W. FAY, M.C., M.B., B.S., F.R.A.C.S. 757

"Some Experiences in the Treatment of Cancer of the Uterine Cervix", by W. G. CUSCADEN, M.D., F.R.C.S.E. 760

"The Treatment of Head Injuries", by ALAN E. LEE, M.D., F.R.C.S. 762

"Pulmonary Tuberculosis, with Particular Reference to Its Early Aspects", by DARCY R. W. COWAN, M.B., B.S. 765

"Dissection or Diathermy of Tonsils?" by E. PAYTEN DARE, M.C., M.B., Ch.M. 771

REPORTS OF CASES—

"Endemic Typhus (Brill's Disease)", by F. BLOIS LAWTON, O.B.E., M.B., B.S., F.R.C.P., and ALAN MURRAY, M.B., B.S. 773

REVIEWS—

Quackery 774

LEADING ARTICLES—

The Gods of Medicine 775

CURRENT COMMENT— PAGE.

Pulmonary Embolism 776

Bronchial Disinfection 777

SPECIAL ARTICLES ON TREATMENT—

The Treatment of Alcoholism 777

BRITISH MEDICAL ASSOCIATION NEWS—

Scientific 779

OBITUARY—

Samuel William Henry Summons 782

CORRESPONDENCE—

An Economic Commentary and Diagnosis 783

CORRIGENDUM 784

BOOKS RECEIVED 784

DIARY FOR THE MONTH 784

MEDICAL APPOINTMENTS VACANT, ETC. 784

MEDICAL APPOINTMENTS: IMPORTANT NOTICE 784

EDITORIAL NOTICES 784

An Address.[1]

By FRANK W. FAY, M.C., M.B., B.S., F.R.A.C.S.,

Retiring President of the Tasmanian Branch of the British Medical Association.

IT is a commonplace of medical philosophy that art is long, life short, judgement difficult and experience fallacious. It would be idle to deny the magnitude of these obstacles that beset the path of the pilgrim to the heights of medical proficiency, but it is not impossible that by taking thought he may journey more pleasantly and rob the obstacles of at least some of their difficulties. To this end I propose to examine this evening a few aspects of professional relationships and allied topics. It would, of course, be an impertinence to lecture to such an audience on the subject of ordinary medical ethics or etiquette, but my desire is to make certain suggestions in regard to matters somewhat outside these—suggestions which, I believe, would, if adopted, promote both professional harmony and efficiency.

Let us consider first the case of Mr. Jones, who has been attacked by pain in the abdomen. He calls in his family doctor, who diagnoses appendicitis and advises operation. Mr. Jones, however, urged thereto by his female relatives, a notoriously suspicious crew who "don't believe in operations", asks for a second opinion. The consultant gives the same advice, and now the trouble begins. The ladies, impressed by the consultant's taste in neckties, insist that he must be the one to operate. He, on the other hand, is naturally unwilling, by yielding to their wishes, to offend the family doctor, who, he knows, is desirous and capable of doing the work himself. On the other hand, if he refuses, he

[1] Delivered at the annual meeting of the Tasmanian Branch of the British Medical Association on March 18, 1933.

knows that the fury of the women scorned may easily result in the dismissal of himself and his colleague and the calling in of a third man; while if, by the exercise of extraordinary tact, he persuades them to let their family doctor perform the operation, it is not very difficult to imagine that the family, if anything goes amiss, will attribute the whole blame for it to the fact that the operation was performed, against their wishes, by the wrong man; and that their chief hobby, for many years at least, will be to damage the reputation of the unfortunate operator and possibly that of the consultant also. All this unpleasantness could, I suggest, be easily avoided if, before the consultant is called in, the family doctor were to arrive at a clear understanding with the patient as to who is to perform the operation.

Frequently, as the result of mere thoughtlessness, we fail to consult our patients' best interests and even give unwitting offence to other professional men. We have a patient whose illness may, we think, be the result of focal sepsis. We examine his teeth—a procedure which, by the way, few of us are fitted to carry out adequately—and then advise him to request his dentist to extract such and such teeth. Naturally even the mildest mannered dentist is annoyed at such an intrusion into his preserves. We can imagine what we would say if a patient came to us and said that we were to give treatment prescribed by a dentist. The dentist rightly thinks that it is his place to decide which teeth are at fault and what treatment is necessary for them. Sometimes we lay ourselves open to still further trouble by putting a patient to the expense of a dental radiograph before he has consulted his dentist. The patient will not thank us if the dentist tells him that the expensive radiograph gives no information that was not obtainable by routine clinical examination. On the other hand, if the dentist very properly prefers his clinical judgement to the evidence of a radiograph, taken perhaps by one with no particular qualifications for the task, we have done him a distinct disservice if our action results in the patient being impressed with a belief in the superiority or even infallibility of X ray diagnosis. I suggest that whenever we suspect dental sepsis to be a cause of ill health, we should refer the patient to his dentist for a report and should avoid comment on his dental condition as far as possible. Moreover it would be an act of courtesy that would, I am sure, be appreciated by our dental colleagues, and would certainly be of benefit to our patients and ourselves if we were to be present when the dentist makes his examination. We should thus learn something of the methods of examination and morbid anatomy of teeth which would be of great practical value, and at the same time we would often be able to give the dentist most important information about the patient's general condition and so enable him to form a better judgement as to whether an attempt should be made to save a particular tooth or not.

It is well recognized that no patient should be given a general anæsthetic until he has been properly examined as to his fitness for one, and that the anæsthetic should be administered under conditions which will permit of the application of the sometimes elaborate restorative measures that may be necessary should anything untoward occur. Yet we are frequently requested to anæsthetize in a dentist's surgery a patient whom we have never seen before. On these occasions neither the time nor the place permits more than a most superficial and perfunctory examination, the patient is fully dressed and often undieted, and it is practically impossible to perform such emergency operations as tracheotomy or cardiac massage or to give adequate treatment for shock. Moreover we frequently find that the dentist proposes to extract so many teeth that the procedure almost amounts to a major operation. Much has been written of late of the danger of septicæmia or some other fatal malady following wholesale tooth extraction at one sitting from patients debilitated from prolonged toxic absorption from dental sepsis, and of the advisability of increasing their immunity by the use of a pathogen-selected vaccine made from a single removed tooth before numerous extractions are attempted. If we were given the opportunity of examining in our own rooms and at our leisure all candidates for general dental anæsthetics, we could determine their fitness to take them and also their suitability to undergo the dental operation proposed. The Council might discuss this with the Dental Association, whose cooperation would be more readily obtained if we assured them that no extra fee would be asked for this service, which is indeed an essential part of the administration of an anæsthetic, and should be carried out as carefully as any other part of it. Moreover we should impress upon the dentists, through their Association, the desirability, in the patient's interest, of all dental operations under ether or chloroform being performed in a private hospital and the absolute necessity of all extensive extractions being made there. Apart from the great discomfort to a patient of recovering from an anæsthetic in a dentist's rooms and of being sent home as soon as possible, and often while still in a state of nausea or shock, in order to get him out of the way—*credite expertis*—the technical difficulties and dangers of an anæsthetic for prolonged extractions are so many and so serious as to demand imperatively that it should be administered only under the best conditions.

It would seem profitable to review also our relations with the radiographer and the pathologist. At the most we send them a few brief lines about the patient's condition when we ask for their assistance. Many of us have got into the habit of professing, almost with an air of pride, that we know nothing about radiology. I submit that this attitude is not creditable to us. Although we may not hope or desire to become expert radiographers, yet a little application would enable us to take

an intelligent interest in the work of our specialist colleagues, and if we were present at the examination we could give the radiographer clinical information that would surely be of the greatest service to him. Questions could be answered as they arise, instead of being left to be asked afterwards and then forgotten. Such a procedure could not fail to be of benefit to all concerned.

Some of us seem to forget that the pathologist is also a medical man. We half resent his desire for clinical information about the former owners of the specimens we send him. An example of the need for cooperation is seen when a request is made for a Widal test. The pathologist cannot properly interpret his results unless he knows the patient's history as regards illnesses and prophylactic injections. We cannot have it both ways. If we want a medical report, we must give him full clinical information. If, however, we want merely a pathological report, I am sure he will be quite willing to tell us the shape and size of the cells, the position and staining properties of the nucleus, the number of the mitochondria *et cetera*, and much good may the knowledge do us.

My suggestion is that since most of us have our consulting rooms within but a short distance of each other, we should take every opportunity of being present at the examinations of patients made by colleagues for our assistance. Such conferences cannot fail to increase our professional knowledge as well as our personal regard for each other.

Often, again, we are guilty of thoughtlessness in our relations with the local pharmacists. The manufacturers, many of them foreign, of proprietary preparations, by constantly bringing their products under our notice, make us familiar with the names of the latter and eventually seduce us into the habit of prescribing them. The evil results of this practice are manifold. The proprietary drugs are almost always more expensive to both patient and pharmacist than their equivalents in the British Pharmacopœia; and, as the pharmacist's profit is generally less on the proprietary substitutes, our gullibility does not increase his goodwill towards us. Many of the costly complex organic proprietary preparations are actually less effective than the much cheaper inorganic salts containing the same element. Moreover, of the pharmacological actions, if any, of many proprietary preparations we have no knowledge beyond that supplied by the manufacturers, who are neither philanthropists nor disinterested scientists. It should also be plain that laymen are just as capable as ourselves of learning the trade names of drugs and, their dosage problems being solved by the handy tablet form of so many preparations, of acquiring the habit of extensive self-medication, the undesirable consequences of which to both patients and doctors it is unnecessary to stress.

The fact is that it should seldom or never be necessary to prescribe proprietary preparations. If any doctor doubts his ability to construct elegant or palatable, inexpensive prescriptions, he may have recourse to the Australian and New Zealand Pharmaceutical Formulary, which has been designed to help him in this very difficulty. This valuable little volume, which contains a suitable prescription for almost every condition in which one is needed, should be the constant companion of every Australian practitioner. If, however, any doctor feels that even with this assistance he cannot overcome his therapeutic laziness or incompetence, then at least he should be patriotic enough to prescribe Empire proprietary preparations, if any be suitable, instead of foreign ones. But if we reduce the local pharmacists to the rôle of mere shopkeepers, we must not complain if we suffer through their profession failing to attract the best type of man.

But we must not forget that even if we resolutely turn our back on the profane territory of proprietary preparations and remain within the sacred bounds of the pharmacopœia, it is still possible for us to commit grave economic sin; and it is an utterly heartless act, particularly in these times of financial depression, to add to the burden of a patient's illness by making him pay for, as examples, an expensive alcoholic tincture which has no advantages over a cheap aqueous liquor of the same drug, or rectified spirits where methylated spirits would be equally serviceable, or a costly and therapeutically inert infusion when inexpensive chloroform water would make an equally effective vehicle. I suggest that the incoming Council carry this matter further by inviting a pharmacist to address the Association on this aspect of prescribing.

I think that it should be more widely known that members of the staff are always willing to demonstrate to any doctor the progress of patients sent by him into the General Hospital. In the past offers to do so have generally been met with indifference and have seldom been accepted. It should be obvious that patients would be bound by closer ties to their family doctor if he showed his continued interest in them by visiting them in hospital, while the consultations with the hospital doctor must inevitably be beneficial to all.

It is becoming increasingly common in other centres for practitioners to form themselves into small groups for study and discussion. Each member of the group engages himself to take a different journal and to write papers based on important articles contained therein. These papers are read and discussed at the weekly or fortnightly meetings of the group. In this way the expense to each man of buying several journals is avoided, and at the same time a very wide field is covered. Those who have adopted this plan have nothing but praise for its results, and I suggest that we might well adopt it in Tasmania. The time and labour involved would be well spent.

It has been frequently pointed out of late that the medical profession does not hold the same place in public esteem that it did, say, a generation ago. Patients in increasing numbers are seeking

the services of quacks of numerous and increasing varieties. Before condemning the stupidity of the public and the unscrupulousness of the quacks, we should make sure that we are free from blame ourselves. It is clear that many sick people get, or think they get, from quacks benefits that we cannot give them. A little reflection will show that the clients of the quacks are chiefly those who need manipulative or psychological treatment. Unfortunately, most of us, before graduation, had little or no training in these branches of therapeutics, and I fear that even now few of us are capable of accurately prescribing or carrying out treatment by massage, electricity, hydrotherapy, or manipulation, or of diagnosing and treating a pure or partial neurosis. It behoves us by recognizing and removing our own deficiencies to make the trade of the quack unprofitable. This we can best do by giving patients certainly and scientifically the relief that quacks give occasionally and by mere chance.

People value very little what costs them very little, and we have not improved our status by our ignoring of this psychological truth. We have numerous clinics, which are attended without fee by many people who are quite able to pay for advice. We should sternly refuse to be exploited by any of these indiscriminate charities, at the same time making clear our traditional willingness to give freely and unstintingly our professional services to the poor. In this way we may help to restore to the community the spirit of independence and self-help that is in grave danger of being lost. This will react in many ways to the national advantage and will certainly help to restore our diminished professional prestige.

And now, after thanking you for your attention, it only remains for me to say how deeply I appreciate the honour of having been your President for the past twelve months. It has been a proud, busy and happy year, and the loyal cooperation of the Council has increased the pleasures and lightened the burdens of the office.

In conclusion, may I take this opportunity of wishing Dr. McIntyre a successful year in the presidential chair?

SOME EXPERIENCES IN THE TREATMENT OF CANCER OF THE UTERINE CERVIX.[1]

By W. G. CUSCADEN, M.D., F.R.C.S.E.,
The Women's Hospital, Melbourne.

TONIGHT I propose to present the statistical record of 105 cases of cancer of the uterine cervix treated by myself at the Women's Hospital.

They extend over a period of three years from July 1, 1929, to June 30, 1932. The majority were treated by radium element (90). The cases are divided into four stages, in accordance with the rules adopted by the Radiological Subcommission of the League of Nations.

[1] Read at a meeting of the Gynæcological Society, Melbourne, on April 4, 1933.

Classification of carcinoma of the *cervix uteri* is undertaken according to the anatomical extent of the growth.

In Stage I the growth is strictly limited to the *cervix uteri*. The uterus is mobile.

In Stage II the lesion spreads out into one or more fornices, with or without extension to the parametrium adjacent to the uterus. The uterus retains some degree of motility.

In Stage III: (*a*) Nodular infiltration of the parametria is present on one or both sides, extending to the wall of the pelvis, with limited mobility of the uterus or massive infiltration of one parametrium with fixation of the uterus. (*b*) There is more or less superficial infiltration of a large part of the vagina, with a mobile uterus. (*c*) Isolated metastases are found in the pelvic glands, with a relatively small primary growth. (*d*) Isolated metastases occur in the lower part of the vagina.

Stage IV comprises: (*a*) Cases with massive infiltration of both parametria extending to the walls of the pelvis. (*b*) Carcinoma involving the bladder or rectum. (*c*) Infiltration of the whole vagina (rigid vaginal passage); or one vaginal wall is infiltrated along its whole length with fixation of the primary growth. (*d*) Remote metastases.

All patients were asked to report six weeks after treatment, every eight weeks for the first six months, every three months for two years, and every six months after that. In spite of all efforts, many patients are lost. Newspaper files are searched and search is made regularly at the office of the Registrar-General of Births and Deaths. The patients who are lost are not all dead; one turned up quite well yesterday (April 3). Of those who died of other causes, one was run over by a motor cycle and one died of cerebral hæmorrhage.

It is unfortunate that so many cases are not diagnosed until too late. Though many cases are only too obvious, there are a certain number in which only a microscopic section can settle the diagnosis.

In every case presented a small piece has been snipped off with a Luc punch. Apart from the fact that failure to examine a snipping renders the statistics useless, in the absence of such an examination cases that were not carcinomata would have been treated and carcinomata would have been missed. With a wide experience I say it is impossible always to diagnose carcinoma without a section. A small, sharp curette will get a small piece out of the cervix in cases of endocervical cancer.

Before treatment blood counts were taken in all the anæmic cases, and one or more blood transfusions were given to any patient showing a low count (under three and a half million red cells per cubic millimetre). Radium treatment given to a patient with any degree of anæmia is useless in all cases and disastrous in many.

In all doubtful cases urea estimations were made and no patients were treated who showed signs of

TABLE I.

Patients Treated with Radium only, from July 1, 1929, to June 30, 1932.

Cancer Group III, 6c. *Carcinoma of Cervix Uteri.*	Number of Cases Treated.	Results of Treatment. Apparently Cured. (1) Alive and Free of Symptoms.	(2) Apparently Cured, but Died of Intercurrent Disease or Injury.	Local Improvement. (3) Improvement.	(4) Local Improvement. Recurrence or Metastases.	(5) Local Improvement. Died from Metastases.	(6) Local Improvement. Died from other Causes.	Not Improved. (7) Not Improved.	(8) Not Improved. Died.	(9) Palliative Relief. Died.	(10) No Information.
Total number of cases treated	90	27		9	3	2	3		31		15
A. Operable ..	12	6 (1 had Radon as well)		1	1				3		1 (Radon as well)
B. Borderline	13	5 (1 had Radon also)		1	1 (Radon also)		1		1		4
C. Inoperable	33	12 (1 Radon also)		5 (1 Radon also)	1	2 (1 Radon also)	1		4		8 (2 Radon also)
D. Very Advanced ..	32	4		2			1		23		2 (Radon also)

TABLE II.

Patients Treated with Radium in Conjunction with Surgery, from July 1, 1929, to June 30, 1932.

Cancer Group III, 6c. *Carcinoma of Cervix Uteri.*	Number of Cases Treated.	Results of Treatment. Apparently Cured. (1) Alive and Free of Symptoms.	(2) Apparently Cured, but Died of Intercurrent Disease or Injury.	Local Improvement. (3) Improvement.	(4) Local Improvement. Recurrence or Metastases.	(5) Local Improvement. Died from Metastases.	(6) Local Improvement. Died from other Causes.	Not Improved. (7) Not Improved.	(8) Not Improved. Died.	(9) Palliative Relief. Died.	(10) No Information.
Total number of cases treated	3	2 (1 had Radon instead)			1						
A. Operable ..	2	2									
B. Borderline											
C. Inoperable	1				1						
D. Very Advanced ..											

TABLE III.

Patients Treated with Radon only, from July 1, 1929, to June 30, 1932.

Cancer Group III, 6c. *Carcinoma of Cervix Uteri.*	Number of Cases Treated.	Results of Treatment. Apparently Cured. (1) Alive and Free of Symptoms.	(2) Apparently Cured, but Died of Intercurrent Disease or Injury.	Local Improvement. (3) Improvement.	(4) Local Improvement. Recurrence or Metastases.	(5) Local Improvement. Died from Metastases.	(6) Local Improvement. Died from other Causes.	Not Improved. (7) Not Improved.	(8) Not Improved. Died.	(9) Palliative Relief. Died.	(10) No Information.
Total number of cases treated	12	4		1		1		1	3		2
A. Operable ..	2	1							1		
B. Borderline	3	3		1		1		1			2
C. Inoperable									2		
D. Very Advanced ..	2										

grave kidney impairment, unless they were first given a week or more of preliminary treatment.

Gross infection of the growth is a very common complication. Douching with carbonate of soda solution and packing with one in one thousand acriflavine solution was used in most cases. Acetone is effective in some instances, but is very painful. A few grossly sloughing growths were associated with a continuous raised temperature, which would not yield to any treatment. These patients were given treatment, in spite of the fever, with, in most cases, immediate and rapid benefit. In these cases the growths are usually very malignant, but very radio-sensitive.

The amount of reaction produced by treatment varies greatly, but very few had very marked reaction. Patients who have been retreated have the most marked reaction, but little can be done for them. Repeated washings of the vagina with low pressure douches were given, care being taken to distend the vagina and to insert the nozzle to the fullest extent to insure that no adhesion took place. The formation of pockets must be always avoided and at subsequent examinations the fine adhesions should be broken down, as can easily be done if too long a period be not left between visits. Ulceration, never a marked feature, became less and less pronounced with experience and care. Dense, hard growths, retreatments, and particularly both in anæmic and debilitated patients, provided the few serious cases.

Proctitis becomes more infrequent with experience. It varies in its severity. In a typical case on proctoscopic examination there is an area of deep congestion with little areas of superficial ulceration. No serious ulceration was noted in this series. Patients with proctitis were given liquid paraffin to keep the motions soft, and were given aspirin, phenacetin and caffeine, with, in a few cases, some *liquor morphinæ hydrochloridi*.

The great majority of patients have been treated with radium element, and for most cases 85 milligrammes of radium have been used. The radium has been distributed, 50 milligrammes in the uterus and 35 milligrammes in the vagina. The radium has been shielded by the equivalent of two and a half millimetres of platinum. The containers are enclosed in rubber tubing. The standard dose has been for one hundred hours. The time has had to be varied with the case, and though most patients have had the whole dose at one time, some have had to have the dose divided, when their condition did not justify the whole dose being given at once.

In the cases treated with emanation, 72 millicuries were given for eight days, shielded by gold containers, the equivalent of two and a half millimetres of platinum. The containers were distributed in the vulcanite plate, one in each of the four tubes, the lead-backed tube being toward the rectum. Extra shielding is provided by the backing to protect the rectum equivalent to one-quarter of an inch (6·0 millimetres) of lead. The remaining containers were placed in the uterus. This made it possible to obtain much more powerful and even fields around the actual growth, with practically no proctitis. No fistulæ formed as the direct result of treatment.

An interesting late result of treatment is the late radium cystitis, which does not come on for at least twelve months after treatment. There were three cases in this series. There are two points of interest about them. First of all, they are apt to be mistaken for recurrence, but the cystoscopic appearances are typical: low, flat, indolent ulceration. The condition is probably due to arteriosclerotic changes in the bladder mucosa. The second point of interest about the condition is in regard to treatment. If the patients are given bladder irrigations with saline solution and are given a saline mixture, the condition clears up. I have, however, seen two cases, not in this series, in which silver nitrate or some stronger solution was used and caused disastrous sloughing. The tissue is already devitalized, and any irritant solution is apt to cause serious damage.

There have been two cases in which the telangiectasis not uncommonly seen in the vagina was present in the rectum, in one case causing hæmorrhage from the bowel. Touching the dilated vessels with the diathermy needle brought about a cure.

Ten patients had radon implanted into the broad ligament for extension into the parametrial tissue. The radon was in nickel containers giving the equivalent of 0·4 millimetre of platinum shielding. One and a half millicuries of emanation were contained in each needle, which was left in for eight days. (An average of about twenty needles was used in each case.) The needles were introduced through the fornices with a long introducer. The same principle was used as in local anæsthesia, the cervix being drawn well over to one side and the introducer inserted into the opposite fornix until it struck the uterine wall. By withdrawing the introducer a varying distance each time, a fan-like distribution can be made in either or both broad ligaments. Three of the ten patients with broad ligament extensions so treated are alive and well.

One patient with sarcoma of the cervix received combined cavitary and interstitial treatment, and is now well after more than three years.

The results of treatment are set out in the accompanying tables.

THE TREATMENT OF HEAD INJURIES.[1]

By ALAN E. LEE, M.D. (Melbourne), F.R.C.S. (England), *Honorary Surgeon to Out-Patients, Brisbane Hospital, Brisbane.*

Factors Accounting for the Special Problems Associated with Head Injuries.

ALTHOUGH the title of this evening's subject has been put down as "The Treatment of Head Injuries",

[1] Read at a meeting of the Surgical Section of the Queensland Branch of the British Medical Association on November 10, 1932.

very little opportunity would exist for debate, or difference of opinion in treatment, were it not that head injuries are frequently associated with cerebral trauma. The enormous mass of literature that has recently accumulated on the subject of cerebral trauma would suggest that this injury offers special problems which are absent in other portions of the body. This is undoubtedly the case, and a special physiological pathology has been shown to exist within the closed cavity of the skull, which modifies to a great extent the normal processes of inflammation and repair following infliction of trauma on living tissue. This special pathology of the brain has been expounded in a masterly manner by Wilfred Trotter, amongst others, and without a knowledge of the peculiarities existing here it is not possible to prescribe any rational treatment of cerebral injury.

The cardinal fact in the physiology of the cerebral structures is that the brain and all its blood vessels are contained within a rigid capsule of bone. Any changes in volume of any one constituent can only be compensated by increased pressure within, or partial exclusion of one of the other constituents. The contents can be classified in three grades: (i) the solid matter of the brain tissue, incompressible in itself; (ii) tissue fluids, including the cerebro-spinal fluid, and any pathological effusions, such as œdema, which follow injuries; (iii) blood, in varying pressures, ranging from the constant high pressure in the arterial system to the low positive pressure in the capillaries, and the varying low pressure, sometimes positive, sometimes negative, in the veins. The cerebro-spinal fluid is under pressure corresponding closely with the venous pressure, and varying with that pressure.

We now have to consider the effects of injury upon these intracranial constituents. When the skull is struck a heavy localized blow, such as when it comes in forcible contact with an unyielding mass, the bony structure of the skull behaves as though it possessed complete elasticity. It undergoes a general deformation, becoming momentarily flattened at the poles struck, and relaxing after the force has relaxed, unless such violence has been sufficiently great to produce a break in the bony contour of the skull. The effect of this sudden change in contour of the skull is to produce: first, a sudden diminution of cerebral capacity; secondly, the infliction of a blow by the bony skull upon the underlying brain at the pole at which the force is applied; thirdly, a corresponding injury by *contre-coup* at the opposite pole. This sudden diminution of cerebral capacity squeezes the brain as though it were a sponge, the effect being distributed throughout the whole cranial cavity, and the fluids, being more mobile, provide the material that is displaced out of the skull. An obliteration of the whole capillary bed will occur, instantaneously producing a widespread cerebral anæmia.

The symptoms of cerebral lesions can be divided into an irritative group and a paralytic group. The irritative symptoms consist in an exaggerated response to stimuli, and can be shown to be evidence of increased excitability of brain tissue. This increased excitability has been experimentally shown to occur wtih various degrees of venous congestion up to complete stasis. The opposite interference with function, namely, anæmia, causes paralysis. Therefore, the sudden widespread cerebral anæmia we are considering will cause a paralysis characterized by instantaneous onset and wide distribution. Seeing that the condition is only momentary in duration, the symptoms will show a tendency to pass off spontaneously as the capillary bed refills. This is the clinical condition known as concussion of the brain.

At the same time, the infliction of a localized blow upon the brain tissue at the pole struck, and its corresponding pole, will produce other effects. Destruction of a certain amount of cerebral tissue will inevitably take place, the amount depending on the severity of the damage. Extravasation of blood of varying extent may be expected, and the development of a localized œdema represents the tissue reaction to the trauma. The production of œdema raises special problems within the skull. There is no room in the skull for œdema; all available space, save a very narrow margin, is needed for the brain and the satisfactory functioning of its blood supply. The tissue fluids increase in volume only at the expense of the circulating blood, and firstly the blood under lowest pressure, namely, that in the veins. The venous circulation is therefore impaired, and venous congestion so produced; the nearer to the œdematous area the greater is such congestion. As I have previously shown, venous congestion means irritation, and the symptoms due to œdema are therefore symptoms of cerebral irritation.

Trotter has pointed out very emphatically that the brain is placed in a uniquely unfavourable position for the recovery from a bruise. The free swelling, which is the normal response to trauma in all parts of the body, can be attained only by interference with the circulation in the region of the bruise. Absorption of œdema is therefore extremely slow, and such an unresolved contusion manifests itself usually, not in the form of local symptoms, though this may occur, but rather as a chronic elevation of intracranial tension, secondary to the chronic elevation of venous pressure. When the condition is severe, there is evidence of continuously raised pressure, while in the milder cases the intracranial space appears to be encroached upon no more than enough to cause undue pressure under special circumstances. The headache that is so common as a sequel to concussion is due to this interference with the mechanisms by which intracranial pressure is normally regulated.

If Trotter's views are correct, the logical conclusion is that the unfavourable outcome of cerebral injury must be due to the unfortunate fact of the closed nature of the overlying skull; and it would appear obvious that the rational treatment of persistent symptoms following cerebral trauma, provided they are of a physical nature and not psycho

genic in origin, should be to convert the skull temporarily into an open container by means of some form of decompression close to the site of damage.

The encroachment upon the available intracranial space, and the persistent raising of intracranial pressure that thereby ensues, is often more evident in cases in which hæmorrhage rather than œdema is the encroaching factor.

An increasing hæmorrhage first takes up the small amount of tissue fluid space made available by forcing the cerebro-spinal fluid into distant portions of the arachnoid system; secondly, it encroaches on the blood supply with lowest pressures, that is, the capillary bed and the veins. Finally, if the increase in volume is sufficiently large, death of cerebral tissue, with atrophy of brain, may occur.

These views of Trotter on the mechanism of head injury are not universally accepted as a full statement of the position, and in recent years certain further considerations have been put forward, especially in the biochemical field, by research workers. Newer work has tended to show that our conception of the cerebro-spinal fluid as a constant volume of fluid primarily designed to protect the delicate cerebro-spinal tissues from injury must be abandoned. These modern workers have shown that the subarachnoid space is the chief fluid reservoir of the body, from which the continually varying water requirements of the circulating blood are drawn.

According to this view, during the various stages of body metabolism there is a constant interchange of blood and reserve fluids, such interchange being determined by the varying osmotic pressure of the blood. Any raising of this osmotic pressure results in an immediate withdrawal of the cerebro-spinal fluid into the blood, and *vice versa*. The fact that such sudden withdrawals of fluid, and sudden changes in tension can occur, must modify our conception of the closed pressure system within the rigid skull. If this view is correct, we see that there are other methods of relieving a general persistent intracranial tension than by surgical decompression. Temple Fay has shown that the most extensive subtemporal decompression must not be expected to enlarge the cranial cavity by a greater extent than ninety cubic centimetres. Relief of a greater extent than this should be readily effected by withdrawal of sufficient cerebro-spinal fluid by changing the osmotic pressure of the blood, and also, it may be said in passing, by repeated lumbar puncture until cerebro-spinal fluid cannot be further obtained.

Still, an additional point of view of the result of persistent, increased intracranial pressure has been put forward by Temple Fay. He likens the cerebro-spinal fluid which encases the brain tissues to a plaster cast encasing a limb. He points out the notorious fact that great care is to be exercised that such a plaster cast does not press unduly on any portion of the limb, or atrophy of the underlying tissues will speedily occur. He states that a persistent increase of intracranial pressure, if unrelieved, in like manner causes atrophy of the underlying brain tissue, and that the tissue most affected is that on the surface of the cerebral cortex, while the well protected vital centres in the middle of the brain stem and pons receive relatively little damage, unless the pressure is very great. He further points out that the tissue of the cortex is the very tissue in which all the intellectual faculties reside, and in which repose all the knowledge acquired by education, and all the faculties necessary for success in the social environment of everyday life. The lack of confidence and general "hang-dog" expression of so many of these victims of severe head injury is therefore to be considered, not as a traumatic neurosis or, worse still, as an aspect of malingering, but as the mental expression of the physical fact of the cortical atrophy due to unrelieved intracranial pressure.

Treatment.

This introductory exposition of the special factors which account for the peculiar problems in head injuries prepares us for a consideration of the treatment to be carried out in such cases in everyday practice.

The first obvious fact in treatment is that there is a vast difference in the degree of severity of different head injuries. The most simple case, in which a patient is admitted to hospital in the recovery stage of a transitory concussion, needs no special therapeutic measures. Such a patient needs only to be put at rest, both mentally and physically, and in a very short time complete restoration of health will occur. The more severely affected patient, whose scalp has been injured, and about whom a history of unconsciousness lasting five minutes or more is obtained, needs special treatment. The first point that matters is that the scalp wound should not be sutured in a routine manner in the casualty room. In many cases the wound communicates with a compound fracture of the skull and perhaps with the underlying cerebral structures. Unless bleeding has to be controlled, only a first aid dressing should be applied before the patient is admitted to hospital. Intelligent care of shock is the first and sole duty of the surgeon after the patient's admission. The application of heat, and possibly the intravenous and subcutaneous administration of saline solution, should be carried out. If a sedative is necessary, it is desirable that morphine should be withheld, because of its well proved tendency to raise intracranial pressure. When the shock factor is safely combated, it should next be determined, by a lumbar puncture examination with pressure readings, whether a raised intracranial tension is present. If, as is usual, such an increased pressure is present, the following procedure should be adopted.

1. Lumbar drainage should be carried out.

2. Dehydration should be encouraged, by limiting the intake of fluids to 900 cubic centimetres (thirty fluid ounces) per day for the early days, and less

still for the few weeks following. The giving of hypertonic solutions, such as magnesium sulphate, by mouth and rectum, and intravenous hypertonic solutions, such as 20% to 50% glucose, or 10% to 15% sodium chloride solution, should be used.

3. Mental and physical rest should be enjoined for three or four weeks, or longer if necessary. A very important measure in reducing intracranial tension is to place the patient in a semi-sitting position. The traditional treatment of the head injury patient, flat in bed without a pillow, has no rational basis in fact.

4. The associated scalp injuries should be dealt with as soon as shock can be controlled.

During the second decade of this century there was, in the United States of America, a great tendency to operate on persons suffering from cerebral injuries, and statistics show that anything up to 25% or more of all severe cerebral injuries were treated by decompressing operations, with a mortality rate approaching 50%. The adoption of more conservative methods during the last few years has produced a corresponding decrease in the mortality.

In my opinion there exist only the following indications for operative treatment. During the first twenty-four hours after the occurrence of a severe head injury, operation should not be undertaken except in the rare case when an extradural hæmatoma increases so rapidly as to endanger life during that period. After the shock period is passed, operative treatment may be carried out: (i) as the treatment of a compound fracture of the skull; (ii) to evacuate a hæmatoma presenting localizing symptoms; (iii) in severe cases of cerebral contusions, which have resisted dehydration treatment. Some weeks or months following the injury, operation may be advisable: (i) to evacuate a chronic subdural hæmatoma; (ii) for the treatment of chronic localized arachnoideal adhesions; (iii) for the relief of convulsions, or for persistent physical symptoms which have resisted long periods of rest and dehydration.

Among those subjects presenting remote symptoms of head injury, at least three distinct types of headache can be recognized.

1. A general dull discomfort, exaggerated by any noise or excitement of any sort; this headache is shared by these persons in common with many other types of psychogenic patients, and appears to be a mental reaction to the physical state of inferiority induced by the cerebral trauma; it is not likely to be relieved by operative measures.

2. Localized headache, splitting or bursting in character, even associated with tenderness in the overlying scalp, close to the site of the primary injury, and usually caused by localized increased intracranial pressure beneath. The increased pressure may be due either to a localized hæmatoma or to arachnoideal cyst formation. The cases in this group are the most favourable for operation.

3. An intermittent headache of a general bursting character, apparently associated with temporary states of hydrocephalus, reacting favourably to lumbar puncture and dehydration methods, and, in the last resort, reacting favourably to operative intervention.

Summary.

To sum up this somewhat meagre discussion of the treatment of head injuries, I would suggest that the majority of those patients who do not die within forty-eight hours of the infliction of the injury, can be ultimately restored to active life by adequate rest and adequate dehydration measures. Of the minority, who become affected with persistent symptoms, appropriate medical and surgical measures can still restore many to a reasonably active existence.

Finally, I would remark that there is every reason to believe that the small minority of persistently incapacitated persons are suffering from physical states which have destroyed the faculties necessary for their economic existence, and it is doubly unjust to class such persons as malingerers or sufferers from "compensation neurosis".

PULMONARY TUBERCULOSIS, WITH PARTICULAR REFERENCE TO ITS EARLY ASPECTS.[1]

By DARCY R. W. COWAN, M.B., B.S. (Adelaide),
Honorary Physician, Adelaide Hospital.

I WISH to thank the President and Council of the Branch for their invitation to speak this evening, and I do so with particular pleasure. For many years I have felt that the importance of the recognition and treatment of tuberculosis in its earliest stages has been insufficiently recognized. I do not exaggerate when I say that the cases of pulmonary tuberculosis I see now are, on an average, in a more advanced stage of the disease than those I saw twenty years ago. This is rather surprising when we consider the greatly improved facilities for diagnosis, particularly on the radiological side, that are available to us today. In the early days of my practice radiological examination of the chest was seldom done. At the present time it is, or should be, done in every suspected case of pulmonary tuberculosis. Practitioners still fail to realize the limitations of the stethoscope, or they refuse to make a diagnosis until tubercle bacilli have been demonstrated in the sputum. Valuable time is lost, and before it is recognized the disease has in many cases advanced to such an extent that the outlook is well nigh hopeless. It is, of course, not always the fault of the practitioner. Usually the disease has an insidious onset and the patient may fail to report until extensive damage has been done. But very often patients do report with indefinite signs and symptoms, and the cause of their vague ill health is overlooked. The methods

[1] Read at a meeting of the South Australian Branch of the British Medical Association on March 30, 1933.

for the control of tuberculosis in this State need tightening up. For too long we have concentrated on the advanced and neglected the early case. The provision of expensive accommodation for patients in the advanced stages without any serious effort being made to get into touch with those in the early stages is foolish and economically unsound. A home for patients with advanced tuberculosis is a necessity if for no other than humanitarian reasons. It is of value also in removing sources of infection. In this connexion, however, it is to be noted that patients are not usually admitted to consumptive homes until they are far advanced, and probably most of the intimate contacts have already been infected. It is the old story of shutting the stable door after the horse has gone. The preventive aspect must be brought more into the foreground, and more serious efforts must be made to recognize the early case and give it adequate treatment.

It is perhaps well to consider for a moment the problem that confronts us. To quote Cumpston:[1]

> In every thousand persons some six hundred or seven hundred are invaded by the tubercle bacillus: of these, two hundred show visible lesions after death, fifteen become clinically identifiable as infected and 0·5 die of pulmonary tuberculosis. The task is to prevent any of the six hundred joining the two hundred, and any of the two hundred joining the fifteen.
>
> At present the vision of a successful tuberculosis campaign is centred too narrowly on the declared case.

This is certainly true in South Australia. The death rates from pulmonary tuberculosis have decreased in every State, but South Australia has the unenviable record of having made less progress than any other State, with one exception. Our climatic conditions are favourable, our population is almost a rural one as compared with some of the other States, there is a notable absence of mining and other unfavourable industries. It seems clear that we have not made the serious effort that we should have to cope with the disease. Holmes,[2] in his excellent report on the control of tuberculosis in Australia, writes:

> In South Australia the control of tuberculosis has not yet been placed on a definite systematic footing. There is little co-ordination in the existing activities relating to the control of tuberculosis.

For many years there have been in this State alone approximately three hundred deaths a year from pulmonary tuberculosis. And what of the living? At the present time the Government has the responsibility for the care of about two hundred and fifty declared cases in hospitals and other institutions, besides many patients undergoing domiciliary treatment and attending the clinic. The cost is heavy and is likely to increase unless we can stem the ever-increasing tide. Owing to the economic distress prevalent at the present time, more and more people are seeking institutional treatment. The need for an organized and sustained effort to combat the disease is becoming urgent. In a campaign of this sort there must be a headquarters and a headquarters staff, and a start must be made at the right end, namely, that of prevention.

Prevention.

Strictly speaking, prevention is the function of the Central and Local Boards of Health, who are charged with the administration of the *Health Act*. But it is not their function alone. A practitioner fails in his duty to his patient and his patient's family if he does not instruct them carefully in necessary precautionary measures. In the control of any infectious disease, notification must be the initial step. South Australia was the first State in the Commonwealth to adopt compulsory notification of pulmonary tuberculosis. The former Chairman of the Central Board of Health recalls this fact with justifiable pride. But the results have been disappointing. This may be due partly to carelessness in the matter of notification on the part of the practitioner concerned. But, to be of value, notification must be followed by effective administrative measures. Something more is required than the occasional visit of a nurse inspector, who leaves a bottle of lysol and a list of instructions, or who disinfects the room when the patient moves or dies. Some local health authorities perform this duty carefully and conscientiously, but others are rather lax. The education of the patient is important, but he is likely to listen to no one so carefully as to his own medical attendant. There must be close and sympathetic cooperation between the health authorities and the private practitioners. Patients will usually obey careful and tactful instruction. But it must be recognized that, no matter how conscientiously these instructions are given and obeyed, infection may pass, if, indeed, it has not already passed at the time of notification, to other members of the household or contacts. The prevention of the spread of infection is the ideal, but probably this ideal will never be completely realized. The next best we can do is to recognize which contacts have been infected and to help them to overcome that infection before dissemination and serious tissue damage have resulted. Herein the boards of health have been working at a disadvantage. It is not their function to make diagnoses, and they have had no encouragement to induce contacts to submit themselves for examination.

The systematic examination and supervision of contacts is an important aspect of any scheme for the better control of this disease. To examine clinically and by X rays and to test every member of a household in which there is a case of open pulmonary tuberculosis means expense, which cannot well be borne by many families even in normal times. But if there were a clinic specially equipped and staffed to carry out this class of work, there should be no insuperable difficulty in getting contacts to present themselves. Nurse inspectors could perform a very useful function if, in addition to their present duties, they were used as a means of inducing contacts to present themselves for examination. Moreover, if a report on home conditions could be furnished to the officer in charge of the clinic, this would furnish further useful information. There is a need for close cooperation

between the Health Department and the Hospitals Department. And if the private practitioner can be pressed into the service, satisfactory results are likely to follow.

Presuming that we will never be completely successful in efforts to prevent the spread of infection, the detection and treatment of that infection in its earliest stages should be our aim. This is a matter that presents considerable difficulties, but with enthusiasm and coordinated effort, a great deal could be done. It is clear that many persons who become infected with the tubercle bacillus throw off the infection and never suffer from actual tuberculous disease. Infection can occur without disease developing, but tuberculous disease cannot occur without infection. The recognition of tuberculous infection is comparatively easy—the tuberculin tests will put us right in this as a rule. But one of our great difficulties is to know which person will overcome the infection unaided or without special treatment, and which is going to develop disease and possibly succumb to it.

Pathology.

In order to have a clear idea of the difficulties we face, it is necessary to study briefly the pathology of early tuberculosis. For tuberculosis to develop there must be infection with the tubercle bacillus. This may gain an entrance to the body either through its external covering (the skin and conjunctiva are rarely the sites of primary foci) or through a lining membrane, especially that of the respiratory or alimentary tracts, or through the tonsil. At the site of inoculation an inflammatory lesion occurs and soon the tubercle bacillus passes to the lymph gland draining that area. This is the simple primary complex of Ranke, and it is important to bear it in mind. A patient whom I saw through the courtesy of Dr. Wunderly illustrates this point so well that the case is worth recalling.

A probationer who was nursing a patient with a discharging tuberculous sinus developed a small inflammatory papule on one of her forearms. She took no notice of this until a few weeks later, when she developed a swollen gland above the elbow. She consulted a surgeon, who removed the gland and had it examined. It contained tuberculous deposits. The surgeon informed me that prior to section of the gland there was nothing about the condition to make him suspect it of being tuberculous. There was nothing characteristic about the primary focus; it was nothing more than a blind pimple, and it ultimately disappeared and left nothing but the smallest scar.

Thus we see, even when the primary focus is on the surface and available for our fullest examination, we may fail to recognize it clinically. How, then, can we hope to do so when it is situated deeply in the thorax or abdomen? There are few, if any, symptoms and no signs, and yet the tubercle bacillus has gained an entrance, and very unpleasant results may follow. The frontier has been crossed, and this may be the beginning of a lifelong struggle for that person. Sir Robert Phillip[3] emphasizes that the proper line is to concentrate attention on the detection of tuberculous infection at the earliest possible moment. "This event can be determined exactly by the tuberculin test, and opens the way to rational and timely employment of preventive and curative measures." He adds: "Having regard to the likelihood of infection in childhood, and the difficulty in assessing the ultimate consequences of such infection, I am firmly convinced that the general use of tuberculin as a diagnostic agent in childhood is not desirable." He advocates routine testing from early infancy onwards. "The procedure", he says, "is painless and harmless, yet the knowledge obtained by it is priceless."

The tuberculin test gives the earliest indication of tuberculous infection when ordinary clinical methods are likely to fail us; but radiography will frequently demonstrate the primary focus in the lung.

The modern conception of the pathology of pulmonary tuberculosis may be summarized somewhat as follows:

There is a primary tuberculous state, following introduction of the tubercle bacillus into the body. This may last for two to ten weeks, and during this time the patient is non-allergic. Complete recovery may occur.

There is a secondary state with rapid development of allergy. In the early secondary state there is a primary focus in the lung parenchyma with involvement of the anatomically related lymph gland. This is the simple primary complex. The primary focus consists of an area of bronchopneumonia, often smaller than a hazel nut in size, and situated in the lung parenchyma, frequently just beneath the pleura. It is most frequent in the right upper lobe, but occurs quite often in the left upper lobe and in both lower lobes. It is least frequent in the right middle lobe. Blacklock,[4] in his informative report for the Medical Research Council, brings out these points clearly.

This state is often accompanied by mild symptoms, such as loss of weight, tiredness, slight fever, pallor, digestive disturbance, and sometimes by cough. The so-called paratuberculous conditions, *erythema nodosum*, phlyctenular conjunctivitis, and pleurisy, are likely to occur, and should at once draw attention to the possibility of tuberculous infection. The Mantoux test gives a positive result and X ray examination may reveal a focus in the lung with enlarged hilar glands. Healing may occur after a variable period, possibly a few months, or the defensive forces may be overcome, the primary focus break down, the lymphatic system become overloaded, and the process pass into the late secondary state, in which dissemination has occurred with the production of miliary tuberculosis or extensive bronchopneumonia and perhaps tuberculous meningitis. This is a grave condition of rapidly advancing tuberculosis and usually ends in death. Serious difficulties in diagnosis are not likely to arise in this stage.

The tertiary state is the common condition of adult phthisis. After recovery from the primary or early secondary state, reinfection may occur. Owing to the degree of tissue immunity that has arisen as a result of the previous infection, the resulting disease is essentially different. It is more localized and chronic, with marked tendency to fibrosis. Aschoff[5] has described this condition well.

The primary infection may occur in childhood, or it may not occur until adult life, when the process, if not limited to the primary complex, is likely to take on the character of the acutely progressing disease of childhood. Similarly, the adult type of chronic fibro-cavernous tuberculosis may occur in a child, but it is rare.

The common conception is that tuberculous infection generally occurs in the early years of life, and that the tuberculous disease of adults is due to reinfection in a person previously immunized. The figures of Heimbeck,[6] using the von Pirquet test, throw doubt on the accuracy of this. They show a comparative minority affected in early childhood, the percentage rising slowly while the patients are kept at home; and when infection occurs at this age it is usually because there is a patient with an open infection at home. With a transition to a working life, the second great period of infection begins, and this probably explains the great morbidity in young adults. Heimbeck's figures tend to show that there is a comparatively small number of persons infected in childhood and that the majority are infected later in life. The morbidity was slight during the childhood period, whereas it was widespread during the young adult period. His investigation into tuberculous infection among nurses was illuminating. At the start of their training all were apparently healthy, but about 50% reacted to the von Pirquet test, and the other 50% did not. There was subsequently a grave morbidity from tuberculous affections among those who originally failed to react, and only a slight morbidity among those who gave a positive reaction. It seems that those persons who contract a benign infection and overcome it are to a considerable extent protected, while those who pass through childhood to early adult life without contracting such infection show a grave morbidity when exposed to infection. It can be said that a von Pirquet reaction in a healthy person may be regarded as a sign of immunity. In other words, an infection which does not reveal its presence with symptoms of disease, but which merely causes allergy, will seldom give rise to disease processes later; on the contrary, it protects the organism against a new exogenous infection. A benign infection with the tubercle bacillus is apparently a good thing, but an infection that leads to the death of nearly three hundred persons per year in South Australia alone can hardly be called a benign one. The haphazard method of allowing people to become infected and working out their own salvation is a dangerous one. If we cannot prevent it, we must make every effort to recognize the presence of infection at the earliest moment and use our best endeavours to limit its possibilities for mischief. The important thing seems to be not so much that infection has occurred, but how that infection is resisted. The von Pirquet test will not help us in this, and reliance must be placed on ordinary clinical acumen and common sense.

If some safe and efficient method of artificially produced protection is discovered, it will be a great step forward. The Calmette-Guérin vaccine is claimed as such a method, but this claim has as yet been hardly established. Heimbeck's experience, when he used this vaccine in an attempt to protect the nurses who started their training with a failure to react to the von Pirquet test, was distinctly favourable. The vaccinated nurses showed a very low morbidity to tuberculosis, whereas similar but unvaccinated nurses showed a high morbidity. He suggests that for persons in close contact with tuberculous patients and failing to give a reaction to the von Pirquet test, subcutaneous vaccination with the *Bacille Calmette-Guérin* vaccine is a valuable method of protection.

From the clinical side, it is not likely that the primary state will be recognized, though recent bacteriological work has shown that even in this stage in some children tubercle bacilli may be demonstrated by the method of gastric lavage and animal inoculation.

But the early secondary state should be recognized. In this stage the Mantoux test gives a positive reaction, symptoms, though indefinite, are usually present, and there may be X ray evidence. Bacteriological work, such as that of Collis and Brockington,[7] makes us alter our views somewhat. We have been in the habit of looking on these primary complex cases as closed lesions; but apparently, even in children, they are frequently open lesions, at any rate during some part of their course. Subsequently, in those who resist the infection well, the lesions heal and become closed again. Seeing that special bacteriological methods are necessary to demonstrate the tubercle bacillus, for practical purposes these children may be looked upon as non-infectious.

It is this early secondary state that is so important. We must realize that symptoms are indefinite and that physical examination as a rule tells us nothing. But the Mantoux test may tell us much, and the radiograph may help in the diagnosis. The ultimate outlook for child or adult depends very largely on the management during this stage. If the condition is recognized and ordinary care is used in treatment, the prognosis is good. The primary lesion tends to heal and recovery occurs with the development of a degree of tissue immunity against subsequent infection. If the condition is unrecognized and untreated, the defensive forces are likely to be overcome and rapid dissemination occur.

Diagnosis.

There are many pitfalls in the diagnosis of early pulmonary tuberculosis. The symptoms are often quite indefinite. They are those of any mild

toxæmia, focal symptoms developing only with spread of the local lesions. Tiredness, loss of weight, loss of appetite and digestive disturbances are perhaps the commonest, but they are lacking in distinction. Physical signs may be completely absent, even in the presence of moderately advanced disease. This point needs stressing strongly. Over and over again I have seen cases missed because no abnormal physical signs were detected. Rivière[8] describes the stethoscope as "a valuable instrument so long as there are no holes in the rubber, no wax in the meatus or prejudice in the mind of the observer; but it is not or should not be the alpha or omega in the diagnosis of pulmonary tuberculosis". The failure to realize the limitations of the stethoscope is probably our most fertile source of error. Assiduous search must be made for the tubercle bacillus; this is the only actual proof of the presence of the disease. But in the early stage there is often no sputum or the search is futile. The finding of the tubercle bacillus means that the lesion is an open one, with all the risks of mixed infection and spread by the air passages. If we allow the diagnosis to rest on the finding of the tubercle bacillus, the patient's best chance of recovery is gone. The radiograph is a very great advance in diagnosis. But it is not always possible to distinguish between shadows due to active and those due to inactive or healed lesions; or to tell with certainty the ætiology of pathological processes that may be disclosed. The tuberculin tests are tests of infection only, and not necessarily of active disease. In a properly carried out skin test the failure to react may, with few exceptions, be taken as a reliable indication of the absence of tuberculous disease either in child or adult. In children, especially in young children, a reaction is of diagnostic value and should not be lightly disregarded. It is of little or no prognostic value, and gives no indication as to whether the person concerned will resist the infection successfully or not. The skin test is harmless and there are no contraindications. A reaction in an adult is of little value as evidence of active disease, seeing that some 50% to 60% of adults react. Even here, however, in a doubtful case, it should put us on our guard. The subcutaneous test is more difficult to carry out, but in the adult is more likely to give us the information that we want, namely, whether there is an active tuberculous process present or not. A focal reaction, that is, a reaction referable to the site of the lesion, in conjunction with a general febrile reaction and a reaction at the site of injection, points strongly to activity of the lesion. The danger of the test has been grossly exaggerated. The one danger to be remembered is the possibility of causing a focal reaction of such severity as to lead to an exacerbation of the disease. This is not likely to occur in careful hands, and if due regard is paid to the contraindications. The test should not be used in the presence of fever, of recent bleeding, or of other severe constitutional disorder. The same danger must be recognized in the therapeutic use of tuberculin. Mild focal reactions imitate Nature's methods and tend to healing; severe focal reactions may lead to breaking down and extension of the disease. Tuberculin is a very powerful weapon, capable of much good in careful hands, capable of much harm in the hands of the careless or ignorant.

All this is rather confusing, but it indicates the necessity of using every method in trying to arrive at a diagnosis rather than relying on one method to the exclusion of others.

A suitable routine in the examination of a suspected case is as follows. At the first visit a careful history of the present and past illnesses is obtained, together with the family history and history of possible contact. A complete physical examination is made, especial attention, of course, being paid to the thorax, and the upper air passages not being neglected. An X ray screen examination is then made. If there is anything suspicious in the findings of the clinical or screen examination, a plate should be taken. Otherwise, for economy's sake, this may be left until the result of a Mantoux test, which should be done next, is known. Sputum, if there is any, should be obtained for examination, and the patient should be told to report in forty-eight hours' time for reading of the Mantoux test. If the results of physical and screen examination and of the Mantoux test are all normal, tuberculosis may be safely excluded. If the result of the Mantoux test is positive, a plate should be taken, as minor changes may easily be missed on screen examination alone. With the information now available one is usually in a position to make a fairly reliable diagnosis. But some cases of chronic non-tuberculous respiratory disease, such as bronchitis or bronchiectasis, are very difficult to elucidate, especially when associated with tuberculin sensitization. In these cases repeated sputum examination must be done. The use of lipiodol is not advisable in the presence of pulmonary tuberculosis. Many patients with bronchiectasis fail to react to a Mantoux test, and this is very helpful in excluding tuberculosis. If doubt still exists, a regular temperature record should be obtained, and in suitable cases a subcutaneous test may be made.

In years gone by great stress was laid on physical signs; but the modern physician will rely much more on X ray examination, which undoubtedly gives more reliable information. We must always keep in mind the fact that pathological changes may be present in the thorax, even to a fairly advanced degree, without causing any abnormal physical signs. In the diagnosis of early tuberculosis we may say that physical signs help us little, if at all.

Some time ago a friend jokingly said to me that I, of course, did not have any deaths from consumption. I sought out my death certificate books, and found that since the war I had signed almost a hundred certificates of death from pulmonary tuberculosis. On looking up the records of these cases, the outstanding fact noted was the advanced state of the disease at the time of its first recog-

nition. Other features were the number of times that such arresting symptoms as pleurisy and hæmoptysis occurred without much notice being taken of them. A great many of these patients had seen doctors. Their symptoms were indefinite and they had been told there was nothing the matter with them, they were anæmic, neurasthenic, run down *et cetera*. In practically every instance the diagnosis was made only when tubercle bacilli were found in the sputum. Altogether it was a rather depressing task and made me realize again how very far we are from reasonable accuracy in the diagnosis of early pulmonary tuberculosis.

Treatment.

It is not proposed to discuss treatment at length. In this there is no royal road and no short cut to success. The main reliance must be placed on the careful observation of the ordinary accepted general lines, which are designed to reinforce natural bodily resistance. In the primary complex stage, probably most patients can be made well by these means alone. But it is our duty to see that the recovery is as complete as possible. The rules to observe are: in active disease with fever from excessive auto-inoculation, rest; if no toxæmia is present, auto-inoculations may be stimulated by exercise, or tuberculin may be used to stimulate reactions. In mild afebrile cases in which there is more or less of a balance between the invading and the defensive forces, tuberculin is useful. It may be given by inunction to children or by injection to adults. Probably its greatest value is in these early cases, in which the resistance is fairly good and the patient afebrile, but nevertheless not progressing quite satisfactorily. It is also valuable in the more chronic type of disease in adults who resist the infection up to a point, but seem unable to overcome it. After a considerable experience of its use I am impressed with the value of tuberculin in such cases. It must not be used in active febrile cases.

"Sanocrysin" is best used in the early exudative types of lesion, and may be used in the presence of fever. Treatment by collapse therapy has no place in a discussion on the earliest stages of pulmonary tuberculosis, but, of course, is a very valuable method in more advanced lesions, especially when unilateral and accompanied by cavitation and hæmoptysis.

Perhaps I may be permitted a few words on the home treatment of pulmonary tuberculosis. In the minds of many practitioners there seem to be only two ideas, that the patient shall be sent north or that he shall be sent to a sanatorium. Before a decision is made concerning what Palfrey calls the placing of the patient, it is well to make up one's mind as to what treatment is likely to be necessary. The extent and the activity of the lesion must be ascertained. As to the former, an X ray examination gives the most reliable information, and for the latter the patient should be put to bed for regular observation of temperature and pulse. This preliminary rest is invaluable to the patient and gives the physician an opportunity of gaining very useful information. Moreover, he can make personal inquiry into home conditions. If these are reasonably satisfactory, I believe most patients do best at home. Sanatoria should be reserved for patients who have no home or whose home conditions are for any reason unsatisfactory. If patients are to be treated at home, it is all the more necessary that they should be instructed in proper precautionary measures and that these should be carefully obeyed. Trouble will rarely arise in a well regulated household. Hospital treatment may be necessary at times during periods of marked activity or for other special reasons.

Be careful in advising patients to go north. Do not send them unless their trouble is a mild one and you are sure they are going to comfortable quarters. There is nothing miraculous in the healing properties of the northern climate, though it is often a useful change for some patients, especially during the winter months. To send a sick patient north to uncomfortable quarters in the middle of summer is a very wrong procedure and may cause much unnecessary suffering. I know of two strong, hardy girls who were born and spent all their lives in Central Australia and who contracted consumption there, apparently from contact with a sick half-caste girl whom they befriended. The northern climate did not protect them. One died and the other came to Adelaide and recovered after a long struggle.

Though plans for a more efficient tuberculosis clinic are completed, we seem to be little nearer to the special department of tuberculosis advocated by Dr. Hone[9] in 1926. A great deal of talk produces little apparent result, and there is a distinct danger that everyone will be tired out before the real work begins. However, if we are instrumental in creating a reasonably efficient organization, we will have accomplished something and can perhaps be content to let the work be carried on by other hands.[1]

Conclusion.

Perhaps it is a policy of perfection to expect the diagnosis of tuberculous infection and its eradication before it reaches the stage of actual disease; but it is on these lines that progress is likely to be made. Let us take a hypothetical case. A probationer in good health and who fails to react to the Mantoux test, is sent to nurse at the Consumptive Home. Three months later she develops *erythema nodosum* (or pleurisy) and the result of the Mantoux test is markedly positive. This must be taken as sufficient evidence that she has been infected with the tubercle bacillus. Would anyone suggest that no notice be taken and that she be allowed to carry on as usual? And yet, unknown to us, this sort of thing is happening every day. A tribute is due to the hardihood of the race that so many resist the infection successfully. But many

[1] At this stage Dr. Cowan showed and commented upon several X ray plates illustrating points he wished to emphasize.

develop actual disease and die of it. Are we as a profession satisfied that we are doing all that is demanded of us? Because we have no direct and certainly no infallible means of combating tuberculous infection or disease, is that any reason why we should ignore it or fail to use to the fullest extent the means we do possess? It is certain that the earlier we diagnose these infections, and the more carefully we treat them, the better results are likely to follow.

References.

(1) J. H. L. Cumpston: "Tuberculosis in Australia", THE MEDICAL JOURNAL OF AUSTRALIA, August 8, 1931, page 153.
(2) M. J. Holmes: "Report on the Control of Tuberculosis in Australia", Commonwealth Department of Health, 1,234, page 2.
(3) Robert Phillip: "The Outlook on Tuberculosis", *The British Medical Journal*, January 10, 1931, page 43.
(4) J. W. S. Blacklock: "Tuberculous Disease in Children", Medical Research Council, Special Report Series No. 172, 1932.
(5) L. Aschoff: "Lectures in Pathology", page 37.
(6) J. Heimbeck: "Tuberculous Infection", *Archives of Internal Medicine*, Volume XLVII, June, 1931, page 901.
(7) W. R. F. Collis and C. Fraser Brockington: "Tuberculosis in Childhood", *The Lancet*, January 21, 1933, page 127.
(8) C. Rivière: "Some Pitfalls in the Diagnosis and Treatment of Pulmonary Tuberculosis", *The British Medical Journal*, September 22, 1923, page 491.
(9) F. S. Hone: "The Prevention of Tuberculosis", THE MEDICAL JOURNAL OF AUSTRALIA, October 23, 1926, page 543.

DISSECTION OR DIATHERMY OF TONSILS?

By E. PAYTEN DARK, M.C., M.B., Ch.M. (Sydney),
Katoomba, New South Wales.

OPINIONS still differ widely about the value of diathermy for the removal of chronically infected tonsils in the adult.

The controversy has raged intermittently for years, but still no decision has been reached, possibly because the great majority of those taking part in it wrote as champions of a pet cause, not as scientists seeking only the truth.

Some of the champions of diathermy appear to me to claim too much: they imply that it is easy, quick, and almost painless. I have been using this method for six years, trying carefully to observe results and to improve my technique, and hope that now I am up to the average in competence. I began with a naïve belief in the claims of the champions mentioned above; after a period of slow disillusionment I have come to the conclusion that to destroy tonsils by diathermy is not easy, quick, and almost always painless, but is slow, difficult, and sometimes fairly painful. But I still believe that it is in most cases essentially the right method, for a reason that has been barely mentioned in all the long correspondence—it never kills the patient.

The protagonists of the dissection method have been just as disingenuous as the champions of diathermy; they always imply that the dissecting operation, in skilled hands, is perfect; they have ignored:

1. Pain and invalidity. These are often of more than two weeks' duration. Some four years ago three patients went from Katoomba to Sydney within a short period; all had their tonsils dissected out by specialists, all had pain amounting to agony, and were laid up in bed for two weeks. One still has painful scar tissue in the fossæ.

2. Remnants. This year I have treated two patients by diathermy for the removal of lymphoid tissue in the tonsillar fossæ; both had their tonsils dissected out by experts.

3. Death. I have personal knowledge of three deaths directly due to the dissection of adult tonsils: two patients died from hæmorrhage and the third from inhalation pneumonia. All were operated on by experts, and all were healthy young adults who should have had many decades of useful life ahead of them.

It is hard to imagine that a conscientious surgeon should consider that he is using the ideal method of removing tonsils when he knows that that method, even in the most expert hands, often causes prolonged agony, sometimes allows remnants of tonsils to remain, and occasionally kills. So great is the power of hunting from consciousness ideas that distress and humiliate, that many throat surgeons may really believe that their patients never suffer much pain after tonsillectomy, and probably very few meet their forgotten remnants; but surely the death of a young and healthy adult will make even a man who allows himself to be called an oto-rhino-laryngologist question whether the dissection method is as perfect as he had believed.

A technique that kills, even most rarely, must have enormous other advantages over an alternative technique that never kills, in order to be rightly considered the method of choice.

To take but one example: Chloroform is far more pleasant than ether, both to give and to receive, but ether has ousted it from its position of standard anæsthetic because chloroform kills just a few more people than ether.

Hoets[1] very necessarily pointed out that it is the surgeon's inescapable duty before every operation to satisfy himself that, at least, it will do no harm. *Primum nil nocere*—many of us have forgotten it in our daily trafficking with pain and death. I think many surgeons pick up the knife with a far lighter heart than they would lie down on the table.

To return to the disadvantages that belong to diathermy:

1. It is difficult. The beginning is easy enough; difficulty starts when the tonsil which is being treated has sunk well behind the anterior pillar. To retract the pillar and apply the diathermy needle accurately to the remnant sounds easy, but often needs much skill and patience. And to be sure that the last speck of lymphoid tissue has gone is also far from easy.

2. It is slow. A large pair of tonsils may need twenty treatments for complete removal. Even if the surgeon has not become weary in well doing towards the end of such a course, the patient may decide that he has had enough.

3. There may be a moderate amount of pain. I have tried three methods of applying the local anæsthetic: (*a*) swabbing the surface at intervals

of five minutes, as recommended by Kent Hughes;[2] (*b*) fixing a cotton wool swab, soaked in the anæsthetic, to the end of a long probe and holding it against the tonsil for ten minutes; (*c*) pressing a cotton wool pad, soaked in the anæsthetic, against the tonsil by means of a special clamp for ten minutes. I have not been able to be certain of perfect anæsthesia with any of these methods, but the last gives the best results and is the most convenient.

In the majority of cases unpleasant after-effects are absent, but sometimes there is slight to moderate pain for two or three days when there has been no burning of the pillars at all, probably due to a slight inflammatory reaction.

4. In a small number of cases the patient will not continue the treatment until all of the tonsils is destroyed. The chief reason for this I believe to be that when the grossly infected part of the tonsil is gone, they cease to have their attacks of "sore throat", their "rheumatism" leaves them, or their general health feels much improved, so, in spite of warning, they go off with a light heart, happily believing themselves "cured". Possibly some of them may be; one could imagine a healthy remnant of tonsil remaining healthy, but the great majority of experienced throat surgeons insist that any remnant is dangerous, and I certainly have not enough experience to begin to think of opposing that teaching, and always consider the work unfinished while there is any tonsil left. On the other hand, the champions of dissection make an unnecessary moan about the difficulty of dealing with remnants; they may be more difficult to dissect than the whole tonsil, but there is no added difficulty for diathermy: it is simply a matter of carrying on from where the previous diathermy (or dissection) left off.

5. Lighting up infection. I have had this happen only once.

> The patient had very large tonsils full of pus; several treatments had been given without causing any pain or discomfort; then, two days after desiccation, there was a violent flare-up which amounted to a very severe attack of acute tonsillitis. This patient declined further diathermy and had her tonsils successfully removed by dissection. Possibly I caused the trouble by trying to go too fast with heavily infected tonsils.

Graham Brown and Duhig[3] have brought forward the most scientific criticism of some of the evil results of incomplete destruction of tonsils by diathermy; but the only general conclusion that can be logically drawn from the article is that diathermy incompetently applied gives poor results. Even if a great many medical practitioners are using diathermy ignorantly and carelessly, that is no condemnation of the method; probably a great many men are using dissection ignorantly and carelessly, and are getting just as bad results, with much greater immediate risks to the patient. This simply calls for teaching that no one should undertake any surgical procedure without getting the knowledge and skill necessary for doing the job properly; and that is practically the conclusion that Graham Brown and Duhig draw: "the authors . . . are satisfied that total removal of the tonsils by electrocoagulation can be performed almost painlessly and with practically no subsequent discomfort . . . provided a little destruction is done at each sitting".

With a further conclusion it is harder to agree—that only an expert throat surgeon can know enough of the anatomy of the part and can have the necessary skill to manipulate the electrode. "In addition, a degree of patience, which is seldom found in patients, and still less in surgeons, is a necessary requirement if the process of destruction is to be carried to its satisfactory conclusion." "Still less in surgeons"—that *plus* natural conservatism probably explains why the great majority of ear, nose and throat specialists can see so little virtue in diathermy for their tonsil work—it is so tedious.

The first half of this accusation is not at all well founded. If the whole procedure is carefully explained to the patient before the treatment is begun, and the necessity of complete removal stressed, and a fixed fee is quoted irrespective of the number of visits, a very great majority will continue to attend as long as it is necessary.

Let us summarize the advantages and disadvantages of each method in competent hands.

DISSECTION.

Advantages.	*Disadvantages.*
Speed.	Severe post-operative pain.
Certainty of complete removal in almost all cases.	General anæsthetic.
	Loss of time, often two weeks.
	Risk of hæmorrhage.
	Risk of inhalation pneumonia.
	Occasional incomplete removal.
	Sometimes death.

DIATHERMY.

Local anæsthesia.	Difficulty.
No hæmorrhage.	Tediousness.
Very little loss of time.	Moderate pain occasionally.
Pain slight or absent.	Possibility of patient not completing treatment.
Certainty of complete removal with correct technique.	Very slight possibility of flare-up of infection.

As both methods hold disadvantages, obviously the perfect way of dealing with infected tonsils in the adult has not yet been evolved, but until the throat surgeon can guarantee that neither pneumonia, nor serious hæmorrhage, nor death can possibly follow dissection, it seems to me that diathermy holds the less serious disadvantages.

The Author's Technique.

The method I now use, after trying several, seems to be the simplest and quite effective.

Employing the monopolar, or Oudin, current, there is no need for an indifferent electrode, and a thick quarter-inch spark gives rapid and adequate desiccation.

With this current one may use a metal tongue depressor and retractor if it is more convenient.

The needle electrode is insulated to within an eighth of an inch of its point by winding round it about four turns of adhesive strapping.

The tonsil is anæsthetized by pressing against it, with a suitable clamp, for ten minutes a piece of cotton wool soaked in Wyatt Wingrave's solution, the formula for which is:

Cocaine hydrochloride, 2·0 grammes (30 grains).
Morphine hydrochloride, 0·3 gramme (5 grains).
Salicylic acid, 0·03 gramme (0·5 grain).
Sodium sulphate, 0·36 gramme (6 grains).
Solution of adrenaline chloride (1 in 1,000), 8·0 cubic centimetres (2 fluid drachms).
Distilled water, 30·0 cubic centimetres (1 fluid ounce).

I was unable to find a satisfactory clamp for my purpose, so had Richard Thomson, of Sydney, make one to my design.

The tonsil having been anæsthetized, the patient is placed sitting in a straight-backed chair in a good light, the tongue is depressed, and the needle is inserted into the tonsil; the current is turned on by stepping on a foot switch. As soon as the tissue round the needle whitens, the foot is raised from the switch, so cutting off the current. This procedure is repeated, with a normal patient, until about half the surface of one tonsil has been desiccated. With a nervous patient, or with an abnormally large or much diseased tonsil, it is wise to do much less, especially at the beginning of treatment. Until by experience one has become confident of knowing how much it is safe to do at a time, it is better to do very little, say, only three or four insertions of the needle.

Each week one tonsil only is treated. Generally, until about half the tonsil has been destroyed, it is not necessary to use a retractor for drawing the anterior pillar of the fauces out of the way, and in some cases a kindly Nature has so designed the parts that the tongue depressor can be used to displace the pillar enough to allow one to attack the last vestige of lymphoid tissue; but in the great majority of cases a retractor is needed in the later stages.

When it has become necessary to retract the pillar, I teach the patient to hold the tongue depressor; I have not yet found one who could not do this satisfactorily, and often with great comfort.

The shape of the entrance to the tonsillar fossa varies immensely. In some cases it is almost circular, with an insignificant anterior pillar; these should be hailed with joy, for the tonsil will be easily accessible, even to the last remnant. In others it is a narrow ellipse, a mere slit, with an aggressively outstanding anterior pillar, and deeply buried tonsil; the prospect, then, is one of difficulty and gloom.

For three or four days after each treatment a gargle of hydrogen peroxide appears to help in the removal of the dead tissue, but it is not essential.

When there seems to be just a thin layer of tonsil left, I place only the point of the needle against it, to avoid too deep a desiccation; or the last stages may be carried through by holding the needle point an eighth of an inch from the tonsil and spraying a shower of sparks upon it.

When no more lymphoid tissue can be found the patient is told to report in a month for further examination; then in three months, and finally in six months, to make as certain as possible that nothing has been overlooked.

References.

(1) J. Hoets: "Diathermy of Tonsils", THE MEDICAL JOURNAL OF AUSTRALIA, September 17, 1932, page 372.
(2) W. Kent Hughes: "Tonsils: Their Surgical Treatment by Diathermy".
(3) R. Graham Brown and J. V. Duhig: "Electro-Coagulation of Tonsils: Some Clinical, Pathological and Bacteriological Observations", THE MEDICAL JOURNAL OF AUSTRALIA, July 2, 1932, page 1.

Reports of Cases.

ENDEMIC TYPHUS (BRILL'S DISEASE).

By F. BLOIS LAWTON, O.B.E., M.B., B.S. (Melbourne), F.R.C.P. (London),
Honorary Physician to In-Patients, Melbourne Hospital,
AND
ALAN MURRAY, M.B., B.S. (Melbourne),
Burwood, Victoria.

SINCE only one case of endemic typhus appears to have been reported in Victoria, it seems desirable to record the present case.[1]

The patient was a farmer aged sixty-four years, living at Tally-Ho, near Burwood. There was nothing in his past history having a bearing on the present illness. It is probable that the disease was contracted in Queensland, for he had spent a fortnight there in a district where other cases had occurred. We do not know whether other cases have occurred in that district recently. The patient left Melbourne on May 22, 1932, and travelled by car through Albury and other inland towns to Sydney, and thence through Newcastle and Armidale to Brisbane, where he arrived on May 29. From Brisbane he went through Moore to Murgon, where he stayed with friends for a fortnight. He visited several places within fifty miles of that centre. On June 15, 1932, he began the return journey, going first to Pomona and Brisbane, and then following coastal roads to Melbourne. He returned to his home on June 28. He was in good health when he left home, but he had a cold when he reached Albury, and he was not well for three days. On his return journey he had another cold and he felt cold and tired during the long stages of this part of the journey.

On June 29, 1932, he ploughed and did general farm work, and on June 30 and July 1 he complained of malaise and muscular pains, but continued working. On July 2 he had headache, and the muscular pains were worse, and he consulted one of us (A.M.).

His temperature was then 38·3° C. (101° F.) and his pulse rate 80 per minute; no abnormal physical signs were found.

During the next two days he was not so well and his temperature was 38·9° C. (102° F.), but no definite signs were observed.

On July 5, 1932, he was much worse, and complained of anorexia, severe headache, pain in the rhomboid muscles, shivering and sweating. The shivering and sweating were worse at night, and he was unable to sleep. On July 6, 1932, a papular rash was seen on his abdomen. There were some stiffness of his neck and a doubtful Kernig's sign. These signs and the severe headache led to a suspicion of meningitis.

On July 7, 1932, the patient was seen by both of us. He complained of severe headache and general pains, but particularly of pain at the back of his neck. He had nausea, but no vomiting, and he had a cough and a little mucoid sputum. His temperature was 38·4° C. (103° F.) and pulse rate 100 per minute. He looked sick;

prostration was marked, and he was flushed and a little cyanosed. His tongue was dry and a little furred, and a definite fœtor was observed, but if there was anything characteristic in this, it was not recognized. The only thing of note in the circulatory system was the slow pulse rate in comparison with the temperature. In the chest there were scattered râles, but no consolidation. The abdomen was distended, but no tenderness or rigidity was found, and the spleen was not palpable. A rash composed of papules and macules was seen on the abdomen. At that time the neck was not stiff and Kernig's sign was not elicited, and lumbar puncture was not done.

Nothing abnormal was found in the urine. The leucocyte count was 10,000 per cubic millimetre, and there was no abnormality in the blood film. A definite diagnosis was not made and a guarded prognosis was given.

Within the next few days it was concluded that the disease was probably endemic typhus, and when the patient was again seen by both of us, blood was taken for Weil-Felix agglutination tests.

When the patient was examined on this occasion (July 11) we felt fairly sure of the diagnosis, and a better prognosis was given although his condition was much worse, and delirium, which was worse at night, had been added to the clinical picture. The headache was still very severe, the sweats were frequent, and the patient was very weak.

On July 15, 1932, improvement was noticed. The temperature began to fall, the rash faded, the headache disappeared, and the appetite returned.

The clinical picture conforms to that described by Adey in other Australian cases, and the agglutination tests support the diagnosis.[2] We wish to express our thanks to Dr. Lucy Bryce, who carried out the laboratory investigations at the Walter and Eliza Hall Research Institute. Her report and graph follow.

Laboratory Investigations.

Dr. Lucy Bryce carried out laboratory investigations with the following results: (i) An attempt at blood culture, made on July 11, was unsuccessful. (ii) From the urine (non-catheter specimen of July 18) a few staphylococci were cultured, but no hæmolytic streptococci. (iii) Serum obtained on July 11 failed to agglutinate *Bacillus typhosus*, *Bacillus paratyphosus A* and *B*, and *Brucella abortus*. (iv) An emulsion of *Bacillus proteus* X 19 was agglutinated by serum of July 11 to a titre of 1/80 after three hours' incubation at 37° C. and of 1/60 after standing at room temperature over night. As this titre was somewhat low to be of certain diagnostic value but was in accord with that often seen at this stage of the illness, three further samples taken at intervals of seven days and a fifth taken on September 12 (some six weeks later) were also examined. These tests demonstrated a rise in titre to 1/320 during the second week of the illness, which was maintained during the third and fourth weeks. The readings obtained by these tests are recorded graphically.

Through the courtesy of the Commonwealth Serum Laboratories, cultures of other proteus strains, "Warsaw", "Kingsbury" and *Bacillus agglutinabilis* U 2 (Wilson) were obtained and tests with the three later samples of the patient's serum were made.

Strain "Warsaw" (indologenous, as was also the original X 19) was agglutinated by higher dilutions than was X 19 (see Figure I).

"Kingsbury" was not agglutinated.

Bacillus agglutinabilis U 2 was agglutinated to a titre of 1/20 after three hours' incubation, but there was some agglutination to 1/80 visible to the naked eye after eighteen hours at room temperature.

With the technique employed the final dilutions of serum used were 1/20, 1/40, 1/80, 1/160, 1/320, 1/640, 1/1,280, made up of equal volumes of diluted serum and bacterial emulsion.

Growths on agar slopes of each of the proteus strains, aged eighteen to twenty hours, were washed off with normal saline solution in amounts which gave the emulsion opacities corresponding to 3,000,000,000 organisms per cubic centimetre. Control tubes containing saline and bacterial emulsion only were set up. Readings were taken after incubation at 37° C. for three hours and after fifteen to twenty hours at room temperature.

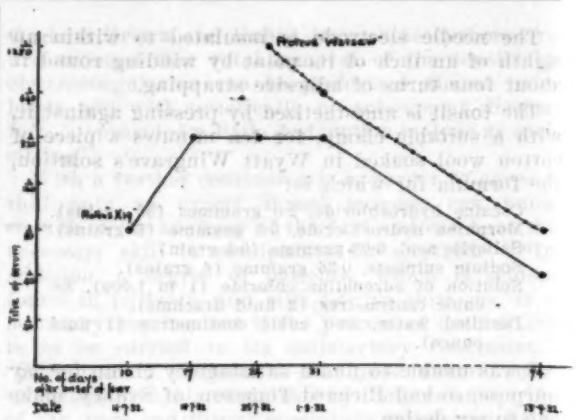

FIGURE I.

Showing agglutination by patient's serum of emulsions of *Bacillus proteus* X 19 and "Warsaw".

References.

(1) W. J. Penfold and A. B. Corkill: "A Case of Typhus-Like Fever", THE MEDICAL JOURNAL OF AUSTRALIA, September 8, 1928, page 304.

(2) C. W. Adey: "Report on Endemic Typhus", Commonwealth of Australia Publication, 1930.

Reviews.

QUACKERY.

THE author of a little book entitled "The Legal and Ethical Aspects of Medical Quackery" is Leonard Le Marchant Minty, a practising barrister who has had experience in appearing for registered practitioners charged with infamous conduct before the Penal Committee of the General Medical Council.[1] The first chapter deals with infamous conduct in a professional respect and is largely an indictment of the General Medical Council and its procedure. The author states that the General Medical Council has no power to swear witnesses and that the defendant has no power to subpœna witnesses. The author states, moreover, that infamous conduct may be construed by the General Medical Council to mean any action which it does not like, provided it is in some degree connected with the practitioner's professional conduct or reputation. He alleges that a book might be written to describe the anomalies, uncertainty and apparent unfairness which may arise out of the exercise of the arbitrary power which the General Medical Council has taken to itself and which the legislature never intended to give it. He also states that the public knows that if a practitioner's name stands high as measured by his income, the Medical Defence Union and the other societies will not have the courage to prefer a charge and the practitioner will continue to write letters to the lay Press, edit popular health periodicals and write under his professional name for them, give testimonials for proprietary remedies and do all the things for which the humbler practitioner would be promptly brought to book.

The author certainly seems to make out a case for a new medical act which will give ample disciplinary powers, but which will, at the same time, provide for them to be exercised under the same rules of natural fairness and justice governing procedure and evidence as adopted in our law courts.

Other chapters deal with bogus practitioners, the herbalist, the bone-setter, the homœopathist, the peripatetic optician and others.

On the whole it is an interesting little book.

[1] "The Legal and Ethical Aspects of Medical Quackery", by L. Le M. Minty, Ph.D., B.Sc., B.Com., LL.B.; 1932. London: William Heinemann (Medical Books) Limited. Crown 8vo., pp. 280. Price: 7s. 6d. net.

The Medical Journal of Australia

SATURDAY, JUNE 24, 1933.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

THE GODS OF MEDICINE.

THERE are many gods in the temple of medicine. Some have been enshrined since the first physician treated the first patient; others have been deified within recent times. There are the gods of truth, of uprightness and of high endeavour. These are placed far above the lesser deities of narrow specialism and have no semblance to the false gods of those who think only of ways and means. The cult of medicine is necessarily polytheistic and its devotee must pay due deference to the several deities; he must seek first the god of truth, the god of uprightness and the god of high endeavour before he bends the knee to lesser gods. Should he fail in his duty, he is likely to lose his professional soul. Witness the slavish devotees of the god of septic foci, those who serve at the altar of the god of diathermy, and those who never leave the precincts of the god of human carpentry, who cures by mutilation. Occasionally a worshipper finds his fellows crowding round the image of one god and utters a protest. Some eighteen months ago an interesting and important discussion took place in the correspondence columns of this journal under the caption "The Worship of the Test Tube". The letter which started this correspondence was a plea for the recognition of the patient "as a human being, and not an enlarged prostate or prolapsed uterus, or even a swarm of septic foci". While not belittling the truly scientific side of medicine, the writer of the letter sought to place laboratory examinations in their proper relationship with clinical examination, not only of the affected system, but of the individual as a whole. The protest was timely. Morris Fishbein gives a devastating description of the "modern medico", whom he describes as the end-product of a perfect civilization. It is worth reading. He has a delightful picture of the way in which the body may be "simonized and interior renovated in thirteen minutes". He adds:

> Heaven grant, friends, that the picture is false, but already there are institutions for the treatment of venereal disease by modern methods where the technique departs but little from this efficiency. The doctor does not know his patients, the patients do not know their doctors . . . . This, colleagues, is a system. One pauses in admiration.

There can be no doubt that if there were no check to the test tube worshipping attitude which prompted the protest of the correspondent to this journal, the practice of medicine would tend towards the systems satirized by Fishbein.

Though, of course, there are exceptions, it must be recognized that medical practitioners as a class tend to get into a groove and to stay there. Unintentionally they see clinical facts and face clinical problems in the light of the system or the method in which they are most interested. Not only is this evident in the habit of taking a short cut to diagnosis by means of the much discussed test tube, but also in the matter of treatment. The submission of patients to surgical operation when operation might be avoided, is too common. Recently, attention was drawn to the advice that cholecystectomy should be practised in the treatment of typhoid carriers, though those who gave this advice admitted that sufficient trial had not been made of the use of the duodenal tube to rid the gall-bladder of the causative organisms. Another and pertinent question has been raised by A. F. Hurst, the senior physician at Guy's Hospital, in an address to the Association of British Surgeons.[1] Hurst points out that in every case of renal disease in which the advisability of operation is being considered, a specimen of urine is obtained by a

[1] *The British Journal of Surgery*, January, 1933.

ureteral catheter and a pyelogram is made. He then refers to the gall-bladder and adds that in the investigation of gall-bladder conditions only the pyelogram is used. He shows that when a specimen of bile is obtained, with due precautions, through a duodenal tube after injection of sulphate of magnesia, information of great value may be obtained in many cases in which a cholecystogram appears to be perfectly normal or in which its interpretation is too uncertain to be of much use. He also states that treatment with the duodenal tube may often make operation unnecessary.

There is no need to labour the points mentioned by Hurst; they have been cited merely as examples. Nor is there need, as might easily be done, to gather similar instances to point a moral. Clinicians must develop a proper perspective. They must leave their grooves and climb to the heights where the whole picture may be viewed. They must follow the example of the late Hamilton Russell, who, we have been reminded, was never satisfied with a difficult method, but laboured till he could find a simpler one. Worship of deities in the temple of medicine is desirable, but devotion must not be centred on one deity alone. Furthermore, devotees must remember that while they follow truth, strive for uprightness and are earnest in high endeavour, their achievement will be greater as they make clinical examination complete and tincture their decisions with common sense; in other words, as they follow simplicity.

Current Comment.

PULMONARY EMBOLISM.

THE lesser grades of infarction of the lung are serious and disabling, and probably many a case of post-operative "pneumonia" has been falsely attributed to cold, to infection, to the baleful effect of anæsthetic vapours and the like, when in reality it followed the liberation of a blood clot into the pulmonary circuit. But these accidents only lead us to the threshold of a much more important and grave happening, the major pulmonary embolism that may cause death so rapidly. Nobody who has witnessed this tragic event in a person apparently perfectly well after operation or labour, or even in the course of a simple attack of superficial venous thrombosis, could fail to carry the ineffaceable impression forever. That a massive clot straddling the bifurcation of the pulmonary artery can rapidly cause death is easy to comprehend. But the exact mechanism is not obvious. G. E. Hall and G. H. Ettinger in describing an experimental study of the subject briefly traverse the historical field, and refer to the classification of Schumacher, who distinguished three types of fatal pulmonary embolism.[1] Of these, one was due to complete blocking of the pulmonary circulation, and caused death rapidly. Another was more delayed, and the fatal termination here followed after some delay, the original thrombus having spread and caused increased obstruction. In the third class only partial obstruction of the artery was found at autopsy, and the exact cause of death was obscure.

The recognition of this last type, together with animal experimentation, has inspired the hypothesis that the plugging of a main pulmonary arterial channel may cause death by giving rise to a reflex inhibition of the heart. Ettinger supposed this to be likely, and assumed that there existed a nerve plexus near the bifurcation of the pulmonary artery which was excited by the distension of the vessel wall as each systolic impulse thrust the clot forcibly onwards. This excitation, he thought, might cause reflex inhibition of the cardiac vagal mechanism or of the respiratory centre. This theory was tested by experimental work.

Dissection demonstrated the presence of nerve twigs extending from the cardiac plexus to the pulmonary artery bifurcation in both dogs and rabbits. This confirmed previous work on the subject, though the nature and function of these nerves was not determined. Next was ascertained the effect of external occlusion by a clamp and also of internal occlusion by clots introduced into the femoral or external jugular veins. As regards the latter, it was found that extensive plugging of the branches of right and left pulmonary artery did not prove fatal unless the clot was large enough to occlude the main pulmonary artery or its orifice. The authors also made blood pressure and respiration records during the experiments in order to determine the effect of the pulmonary occlusion on the circulation and respiration. In addition, they tried the effect of stimulation and also distension of the pulmonary artery wall.

In discussing their results, they point out that if death in man does occur in cases of partial obstruction of one branch only (Schumacher's third type) it does not parallel the experimental findings in animals. They remark that possibly there may be some reflex phenomenon that does not occur in dogs, or that autopsies have failed to demonstrate a clot in the right ventricle plugging the pulmonary artery orifice. In no case did they obtain any evidence of reflex disturbance in animals of the heart or medullary centres by increasing pulmonary arterial pressure even to four times the normal. Aortic blood pressure, of course, fell in these experiments; this naturally follows the inevitable blood starvation. Hall and Ettinger suggest that any element

[1] *The Canadian Medical Association Journal*, April, 1933.

of "shock" is really due to medullary anæmia. They also remark that some of those persons dying from pulmonary embolism are past middle age, with presumably some degeneration of the cardiac muscle, and it is possible that right ventricular failure could readily occur in such persons. The failure of these experiments to produce any reflex inhibition of the heart by clamping the pulmonary artery is also significant. Hence the work failed to support the hypothesis of reflex inhibition or shock. Certainly human beings often survive very large pulmonary infarctions, but these are probably not produced very rapidly. When massive obstruction occurs, of course, the circulation cannot go on. It still may be possible that reflex inhibition may occur in man, though the animal work does not favour this theory. But the important thing to remember is this, that the risk in clinical pulmonary embolism is probably mechanical. No treatment designed to alleviate shock can do the slightest good when the main vessels to the lung are occluded. So we appear to be back to the surgical suggestion of Trendelenburg as regards treatment—heroic, but perhaps justified if the man and the equipment are immediately available.

But in the midst of these suggestions, ranging from the academic physiological to the dramatic surgical, let us not lose sight of prophylaxis. The clot is supposed usually to arise from one of the larger venous "pools", like the iliac veins, and our duty is to avoid those extremes of post-operative and *post partum* treatment that are conducive to stasis. Fortunately, this is usually done today. The patient is not immobilized, and a more vigorous circulatory function is encouraged, especially as regards venous return. It does not seem possible to eliminate all possibility of this rare but dreadful catastrophe, but the understanding that at least it seems to require the presence of a large clot may help to lessen its occurrence.

BRONCHIAL DISINFECTION.

WHEN Lister revolutionized surgical practice, the repercussions of his discoveries upon the therapeusis of medical diseases could hardly have been foreseen. But with the striking advances of bacteriology have come many efforts to attempt the same disinfection of infected tracts of the body as the Listerian method accomplished for the surgical wound. True, the parallel is not exact, for surgery aims primarily at the exclusion of infection, but the experiences of the world war showed us that antiseptic treatment still held its place in the handling of bacterially contaminated tissue. Persistent and sincere have been many of the efforts to effect partial or complete sterilization of the inner surfaces of the hollow viscera when attacked by acute or chronic infective processes. But the successes have been few. Blood disinfection has proved illusory, so also has that vainest of hopes, the disinfection of the lower alimentary tract by drugs given by mouth. Local applications have met with partial success. Irrigation of the colon, the bladder, the pelvis of the kidney, and even the subarachnoid space has given some grounds for encouragement. The latest in the field is bronchial disinfection, and review of this is made by John A. Kolmer, who uses the experimental method in animals to obtain his data.[1] Kolmer regards the streptococcus as the most important infective agent attacking the bronchial tree, other primary infectors being the staphylococcus and pneumococcus, the spirochætes being secondary invaders.

Kolmer lays down that if bronchial disinfection is possible the bactericidal agent must be active and rapid, of high penetrability, feebly toxic and free of injurious effects upon the pulmonary tissues, such as inflammation, lowering of reflex excitability, and production of œdema. He further points out that a suitable substance would need to be applied frequently, even daily, to produce much effect. He therefore has compared the experimental bactericidal properties of a number of antiseptics with their degrees of toxicity and capacity to inflict local damage when introduced frequently into the bronchial tree of rabbits. Injections were given every two days to the animals, the amountz used being comparable with those suitable for irrigation of the bronchial tree of the human subject. The organs, especially the kidneys, were examined for signs of spoiling, and the bronchial mucosa was histologically examined.

Kolmer arrives at the conclusion that the bronchi of human beings could be safely irrigated several times weekly with successive amounts of 15 cubic centimetres of fluid, making a total of 250 cubic centimetres for each lavage, 15 cubic centimetres being left in at the conclusion. The solutions he believes safe include such substances as the dyes, mercurophen or metaphen, potassium permanganate, gomenol, chloramine T, and neoarsphenamine. He recommends a menstruum containing sodium, potassium and calcium chlorides where possible.

This work has been inspired by the modern work of the expert bronchoscopist. Obviously it has a very limited application. But where skilled hands are available bronchoscopic drainage is of known value in cases of chronic pulmonary suppuration, and it is interesting to know that a reasonably active disinfecting fluid may be used for lavage of the air passages and even left in the lungs without damage.

Special Articles on Treatment.

(Contributed by request.)

XI.

THE TREATMENT OF ALCOHOLISM.

As analytical psychology gives us a deeper insight into the personality of the individual, many of the now accepted views upon the part played by alcohol in the production of psychotic states will require modification.

[1] *Archives of Internal Medicine*, April, 1933.

It is a mistake to regard alcohol as an ætiological factor in every psychotic case in which there is a history of previous indulgence. Alcohol, in fact, may just as often play a precipitating part, as it does in some cases seem to be the exciting cause.

The precise effect of large quantities of alcohol upon the human organism is not yet clearly understood. Its effect upon somatic structure is variable, but whether it acts chiefly as a direct poison or secondarily through the disturbance of metabolism incidental to continued indulgence, is a matter which for the present remains undecided. There is no uniformity in the reactions of the human organism to the taking of alcohol.

The vulnerability of specific organs to toxic agents is a subject still shrouded in ignorance, although it is assumed that behind the so-called alcoholic insanities lies either an inherited psychopathic taint or a diminished neuronic resistance to the drug. Even here the reactions differ, for mental alienation of alcoholic origin presents symptoms of every possible variation.

Apart from the inherent psychopathy, underlying emotional difficulty or constitutional inferiority which may give rise to over-indulgence in alcohol, it is essential to determine the type of mental disturbance associated with alcoholism before essaying treatment. Alcoholic cases may present great difficulty in diagnosis, because over-indulgence is sometimes a symptom of a psychotic state having no direct ætiological relationship to alcoholism. It is often a symptom in early melancholia and in the expansive phase of general paralysis. It may occur in the prepsychotic stages of *dementia præcox*, or may mask an essential epilepsy. And unless one can differentiate clearly between those mental states in which alcoholic indulgence is an incidental symptom and those in which it seems to play a specific part, treatment may be misapplied and its effect become misleading.

Three Types of Drinkers.

There are, roughly speaking, three types of drinkers: (i) Those to whom the taking of alcohol is part of the social custom—those who drink only in company and for the sake of conviviality, but who do not drink by themselves and in whom there appears to be no craving. (ii) Those whose drinking is done periodically and who are at other times total abstainers; these are classed as dipsomaniacs, and they frequently show some nervous instability, a cyclothymic temperament or a recurrent subconscious homosexual trend. (iii) Those who habitually abuse alcohol; the chronic alcoholics to whom, as the result of some temperamental defect, alcohol has become a neurotic need. In Types (i) and (ii) there sometimes develop acute psychotic manifestations, such as acute alcoholic poisoning, alcoholic hallucinosis, paranoid episodes or *delirium tremens*. In Type (iii) the continued abuse of alcohol may give rise to any of several of the chronic alcoholic psychotic states, such as Korsakow's psychosis, chronic alcoholic hallucinosis, simple alcoholic deterioration and alcoholic dementia. But, apart altogether from the end results of excessive drinking, whether they be *delirium tremens* and death or Korsakow's psychosis and dementia, the continual taking of alcohol or the periodic debauch is in itself a symptom of a personality disorder which is generally the underlying cause of the whole trouble. The cure of alcoholism is therefore twofold. It is not enough to bring about a physical weaning from distilled spirit or fermented liquor. It is not enough to reestablish regular sleep and promote an adequate appetite. The correct and basic treatment of alcoholism aims at removing the cause. Alcohol is a psychological crutch, and the patient cannot be cured merely by taking away the crutch. The successful physician must seek to heal the mental cripple by implanting a new technique of living, by helping his patient to develop a better adjustment to reality, to overcome his feelings of frustration, and attain an attitude of affirmation. This, needless to say, is the more important part of the cure.

Where to Treat the Patient.

It is very necessary, before treatment is commenced, to determine where the patient is to be nursed, and the doctor's decision will be governed chiefly by the type and severity of the disorder which is presented. That the alcoholic should not be nursed at home is almost axiomatic. It may be necessary to have him certified as insane and delegate his treatment to the medical staff at a mental hospital. Patients with acute alcoholic poisoning and those with mild *delirium tremens* may be treated in a private or public hospital. But patients with dipsomania and chronic alcoholism should, in many cases, be certified under the *Inebriates Act* and sent to the special State institutions, unless the medical practitioner can secure the confidence and complete cooperation of the patient. The mistake most often made is in not segregating the patient or not keeping him segregated long enough. It requires scarcely any skill to wean the average alcoholic of his drug and administer the appropriate supporting treatment; but it frequently taxes one's judgement and calls for wide experience to know how long a particular patient should be kept segregated or under strict observation.

How to Treat the Patient.

Treatment must in all cases be modified to the particular needs of each individual patient. It consists of employing the best suited methods of combating the toxic effects of the drug while at the same time augmenting the patient's strength and allaying psychomotor restlessness.

Two acute alcoholic conditions call for both prompt and adequate treatment: acute alcoholic poisoning and *delirium tremens*. In both cases the patient is acutely ill. The danger point may be quickly reached and death may supervene with devastating suddenness.

Acute alcoholic narcosis follows the consumption of large quantities of liquor, from which the patient sinks into a state of coma. The patient frequently regains consciousness within a few hours, even in the absence of treatment, but sometimes, especially when the condition supervenes upon renal or cardio-vascular disease, the coma deepens and the patient dies from respiratory failure. The treatment consists of washing out the stomach as soon as possible with warm water and administering warmth and the appropriate cardiac and respiratory stimulants.

Delirium tremens may follow upon inordinate indulgence, acute infection or traumatism occurring in an alcoholic. It is a current and widely accepted belief that this condition may result from the withdrawal of alcohol. But, whatever the cause, there develops an acute hallucinatory delirium with marked toxic symptoms, psychomotor restlessness and tremor. Here, as in other alcoholic states, the details of treatment must be adjusted to meet a divergence of symptoms. Basically, the treatment consists in supporting the patient's strength, the timely and proper administration of food, the withdrawal of alcohol, the allaying of restlessness and insomnia, dehydration and the elimination of toxic products by free catharsis. Food must be adequate in amount and of high caloric value, whether it is administered by a nasal tube or by a tactful and patient nurse. The giving of food must be pushed to the limit of the patient's assimilative powers. It is not always necessary to resort to tube feeding, but this method should on no account be withheld if one experiences any difficulty in persuading the patient to take nourishment. It has an added advantage in that appropriate sedatives, purgatives and stimulants may be given at the same time. It is unwise to use sedative drugs if rest can be obtained by prolonged hot baths or by the use of the cold pack. There is no specific sedative, but if a quick-acting sedative is required, paraldehyde, given in drachm doses every quarter of an hour until the patient sleeps, provides a safe and comparatively easy method of sedatizing the alcoholic. When it is necessary, in cases of extreme restlessness, to use hyoscine and morphine hypodermically, they should be given in large doses. Apomorphine is occasionally used, but the dose should not be large enough to produce prolonged or violent emesis, nor should it be given to prostrated patients. The bowels must be freely opened either by purgatives or bowel wash-outs, and the bladder should be catheterized if there is a tendency to distension. Diaphoresis obtained by means of packs or hot air baths is a valuable adjunct

to other methods of detoxification. In my own experience lumbar puncture, by dehydrating the cerebral tissues, tends to allay the restlessness and promote sleep.

Some types of *delirium tremens* appear to resist all the ordinary forms of treatment; the patient rapidly becomes prostrated and develops a low muttering delirium which is inevitably fatal. In such cases, I believe, repeated spinal drainage, in addition to the usual treatment, offers the only hope of saving the patient's life.

Acute and Chronic Alcoholic Hallucinosis.

Though of much graver import from the mental viewpoint, acute alcoholic hallucinosis bears a strong resemblance to *delirium tremens*. Acute auditory hallucinations dominate the symptom-complex, and aid in the formation of a delusional system. The physical condition of the patient is seldom alarming, and in consequence the treatment is carried out under custody in order to minimize the risks from violence. The patient should be certified and nursed under the safeguards of a mental hospital. Likewise with the chronic alcoholic hallucinoses and delusional states which develop in the susceptible after prolonged indulgence. Little can be done to cure the patient whose symptoms reflect a toxic spoiling of cortical neurones. Custodial care, together with such symptomatic treatment as the physical state demands, is rendered necessary, and the best that can be anticipated in the majority of cases is that the patient may become adjusted to the routine of the hospital and find useful occupation in some simple productive task.

Korsakow's psychosis is a mental disorder of toxic origin associated with peripheral neuritis. Alcohol is by far the most frequent cause. The patient exhibits disorientation, fabrication, loss of memory for recent events, with suggestibility and hallucinations. The polyneuritis may be mild, producing only muscular weakness, diminished reflexes and pain over the main nerve trunks, or it may be intense with prostration, muscular incoordination and paralysis. Treatment of the physical symptoms, in addition to the withdrawal of alcohol, is along the accepted orthopædic lines. Absolute rest must be secured, and careful attention must be given to the detoxification and support of the patient's strength. The question of certification and treatment in a mental hospital can be settled only by a complete survey of the symptoms, and especially the mental symptoms, for many patients exhibiting this disorder will require custodial care for the remainder of their lives.

Permanent institutional care is the only treatment for cases of alcoholic dementia. This is the terminal stage of the majority of the alcoholic psychoses, but a somewhat similar condition, known as alcoholic deterioration, may develop insidiously in chronic drinkers who may never previously have developed psychotic states. In this condition there is a general failure of all the mental faculties, first noticed in the ethical and emotional spheres, but gradually spreading and extending to a total personality slump, immune to any known form of therapy.

Little need be said about the treatment of such conditions as alcoholic pseudoparesis, alcoholic epilepsy or alcoholic pseudo-paranoia, as they are all different varieties of chronic alcoholic insanity, and from a therapeutic point of view only possess an "asylum interest". Certification and committal to a mental hospital is practically the only course open to the medical practitioner should any such cases be encountered in private practice.

Psychotherapy.

As already indicated, there are certain alcoholic conditions which cannot be cured, and other conditions in which alcoholism is primarily a symptom of an underlying psychosis. Addiction to alcohol is in itself the outcome of a psychological maladjustment, the treatment of which forms the chief part of any radical cure. Having treated the physical effects of alcohol in a patient who has become addicted to its use, the aim of the physician should be to secure total abstinence, for such a state forms the only reliable prophylaxis in the prepsychotic period in persons of neuropathic make-up or of insane heredity.

Alcoholics display an infantile attitude to life. They are unable to face reality and to share in the dividends of maturity. Fearing life, they find courage in cognac, or relief in rum. Fixations and regressions due to faulty upbringing and lack of parental guidance, together with adverse environmental states, may lead to chronic alcoholism. Mental conflicts, fears, anxieties and other neurotic handicaps form the chief figures in the ætiological background of the alcoholic. Anything, in fact, which hinders the individual from reaching psychological maturity may give rise to the alcoholic habit. It therefore falls to the lot of the physician to understand these neurotic tendencies so that he may give insight to his patient and bring about a better adjustment to reality by the explanation of these psychological difficulties.

The psychotherapy of the alcoholic is often a tedious business and requires much time, energy and understanding on the part of the physician. It is futile to imagine that simply by shutting a person up for a certain length of time his "craving" for alcohol will disappear. It will not disappear until the conflict out of which it arose has been solved by the physician, and the patient has undergone some form of psychological analysis and reeducation. This can be brought about only after one has gained the confidence and cooperation of the patient, and it is often a difficult task to persuade the patient to surrender his preoccupation with the hedonistic sideshows of the alcoholic life and stand four-square to reality. There are some patients who are constitutionally incapable of accepting a stalwart and adventurous attitude to life's hazards, and for them it would seem that some form of permanent segregation offers the best solution to the trouble. For others there is often a much happier outlook if the physician has the knowledge and sympathy to guide them through the wilderness of despair to the promised land of health and mental poise. It is indeed the bounden duty of the physician to inculcate in his patient ideas of right living and right thinking, to understand mental mechanisms so that he can put courage in the place of timidity, banish fear, erase the sense of guilt and make reality appear more desirable than dreams, always remembering that chronic alcoholism is the expression of frustration and that suicide lurks in the shadow of failure.

REG. S. ELLERY, M.D.,
Honorary Psychiatrist to the Alfred Hospital and Consulting Alienist to the Women's Hospital, Melbourne.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Anatomy Lecture Theatre, University of Adelaide, on March 30, 1933, DR. ST. J. POOLE, the President, in the chair.

Pulmonary Tuberculosis.

DR. DARCY R. W. COWAN read a paper entitled: "Pulmonary Tuberculosis, with Particular Reference to Its Early Aspects" (see page 765).

DR. FRANK S. HONE said that Dr. Cowan deserved the thanks of all present for bringing so important a subject before them in his usual clear, bright and vigorous manner. His utterance was particularly opportune. He had referred to the lack of coordinated effort in the past against pulmonary tuberculosis in South Australia. Thanks to the cooperation of the Inspector-General of Hospitals and of the Chairman of the Central Board of Health with a committee of the Branch during the past year, there seemed a reasonable hope in the near future of better directed effort than they had ever had. If that hope were realised, the responsibility would rest with them as individual practising members of taking their share in the campaign. Increasing experience convinced Dr. Hone

more and more that hope of future improvement lay only in the campaign. Additions to knowledge during the past thirty years had given them the right to ask for this, and if the opportunity was now given them, they would be to blame as a profession if no improvement in their figures took place in the next ten years. For that reason Dr. Hone was grateful for the privilege of opening the discussion and in what he said he desired to emphasize the part the individual practitioner might play in prevention.

At first sight it might seem surprising that Dr. Cowan should introduce a paper on the early diagnosis of pulmonary tuberculosis by a discussion of the number of late open cases. Yet this was the only way to emphasize the extent of the problem and it was only as they realized its extent that they perceived its seriousness. The task of early diagnosis was so difficult in individual cases that they would not be prepared in every case to face up to the difficulty unless they realized its seriousness. They had to educate their medical conscience till they regarded it as just as serious a blunder to allow a closed case of pulmonary tuberculosis to become an open case as to allow a closed case of tuberculous hip disease to become an open case. Dr. Hone showed a graph to demonstrate Dr. Cowan's point that the mortality in South Australia, though falling, had not fallen as rapidly as in the rest of Australia. The chart of figures showed that their efforts at prevention so far had mainly resulted only in the prolongation of life of individual sufferers, but had had no appreciable effect in reducing the number of fresh infections. Granted that the rise in the last column was, to a certain extent, due to the greater activities of the Central Board of Health, especially since Dr. Southwood became Chairman, this did not wholly explain the steady rise for the past ten years. And probably it did not show the whole picture. For Sir Robert Phillip estimated before the war that for every death, there were three patients living with active tuberculosis and during the last few years he had altered this to nine patients living, so that the true number of active cases was ten times the number of deaths. This would make their active cases 2,500 to 3,000. Even if they took the lower figure of 1,000, the important point for them as a profession was that with only 250 beds available in institutions for these patients, 75% of them must be in the care of individual members of the Branch. The greatest responsibility for the prevention of fresh cases arising from them, therefore, rested with those who were in active practice; for every fresh open case arose from one of these patients.

The problem was much the same as for other diseases spread by direct infection. The curves for diphtheria, for instance, showed the same persistence in the number of cases, but prolongation of life by better treatment. In this disease they had supplemented previous methods by immunization of those exposed to infection. As Dr. Cowan had pointed out, they could not, as yet, do this with pulmonary tuberculosis. The disaster with *Bacille Calmette-Guérin* in Germany last year had been proved to be due to contamination and had no more to do with the value of the vaccine than the Bundaberg disaster had to do with the value of prophylactic immunization against diphtheria. But the case for *Bacille Calmette-Guérin* at present could not be compared with the case for antitoxin and until they had more evidence they had no right to use it.

They were driven then to early diagnosis of infection in an endeavour to prevent this infection developing into disease, and certainly into an open case and, therefore, infective. And from his own experience Dr. Hone realized that the repeated careful examinations and thought necessary for investigation implied a very high morale on the part of men engaged in busy lodge practice.

They had four means at their disposal: history, observation, radiology and laboratory methods. While Dr. Cowan rightly emphasized the value of radiography, Dr. Hone was sure he did not mean them to depend unduly on any of them. Any one of them by itself might prove fallacious, and its undue exaltation in their practice might land them in disaster. They needed them all and they needed also a great deal of common sense and calm judgement rightly to appraise the value of each in a difficult case. There were two points of supreme importance in history. The first was information as to any recent acute resepiratory infection or succession of infections, or of recent overwork, injury, lactation, or similar exhausting conditions, for any of these provided the opportunity of endogenous reinfection from a primary focus which pushed the patient into the exudative stage. The second was a history of old or recent close and repeated contact with an active case, for this provided the opportunity of exogenous reinfection, with the same result. Dr. Cowan had not made it quite clear to Dr. Hone which of them he was emphasizing when he spoke of reinfections. If they kept their eyes open, they saw how each played its part, and when both factors came into play, as in overworked domestic servants in a house with a patient in the terminal stages of the disease, or waitresses working for long hours in overheated and overcrowded restaurants, Dr. Hone proceeded with the utmost caution before ruling out the possibility of early tuberculosis.

In children, of course, family contact was of supreme importance. The figures obtained by Dr. E. Britten Jones in the routine examination of children for the past two years at the out-patient department of the Adelaide Children's Hospital had confirmed experience elsewhere. His figures showed that with the routine Mantoux test children with no history of family contact gave 10% positive and 90% no reaction, but in those with a history of definite family contact he obtained 60% positive and 40% no reaction. On the other hand, in 130 cases of children who were similarly tested, there were 40% positive and 60% who did not reast; this suggested that these had had greater care.

Dr. Hone had been thinking during the past few days how hard it was for them to realize all the implications of fresh knowledge on a fresh subject with which they were familiar. A radium clinic was a new thing, and in every clinic in Australia the nurses must have their blood examined once in three months, yet Dr. Hone did not know that at any tuberculosis clinic or hospital in Australia periodical X ray examinations of the chests of its nurses were made or intracutaneous tuberculin tests carried out.

Dr. Cowan rightly deprecated undue reliance on negative findings with the stethoscope. German writers now said: "Early tuberculosis is seen, not heard", thus supporting Dr. Cowan's argument for radiographs rather than auscultation. Yet in a patient who was fortunate enough to have an attack of hæmoptysis early in the exudative stage, one might detect physical signs. And they would err if they omitted other forms of observation: regular taking of evening temperature for a longer or shorter period, regular weighing, quite apart from clinical examination of the chest and of other organs for signs of trouble elsewhere.

Dr. Hone did not know that he could go as far as those German authorities who said that it was unethical to say the lungs were healthy without making an X ray examination of the chest, but Dr. Hone did agree most heartily with what Dr. Cowan said about the value of X ray examination in any doubtful case. Comparing his last five years with the previous five years, Dr. Hone realized how much more frequently he employed this method of investigation than he had done, simply because of his increasing satisfaction with results. He did not share Dr. Cowan's enthusiasm for screening. On the contrary he had given it up some years previously, because in these very earliest cases, which were their chief anxiety, he found he was missing shadows which a film revealed; and conversations with radiologists had confirmed him in this view. What he found of most value was periodic radiographic examination at intervals, because he had then a pictorial record, more permanent than his notes or remembrance of breath sounds, or adventitious sounds. He had found this of special value in patients proceeding to other States or other countries. The X ray films of their chests were much more valuable than the photographs on their passports.

There were two points that Dr. Hone would like discussed. His own opinion was that a general practitioner who took his films, unless he had a fair amount of experience, ought to be very careful about expressing a positive

opinion without a consultation with a radiologist. During the past few years Dr. Hone had had two or three women come to him in great anxiety some two or three years after they had been told they had early pulmonary tuberculosis on the strength of an X ray examination made by their medical attendant. In these cases the subsequent history was against the truth of such a diagnosis, and Dr. Hone could find no clinical evidence of disease, nor did the X ray pictures shown to him suggest it.

For that reason he preferred his skiagrams to be taken by someone who specialized in this work, and he conferred with the radiologist on the interpretation of the shadows he found in conjunction with the other information he (Dr. Hone) had obtained. In this way each of them was constantly learning, even by their mistakes.

In the past the question of expense had often been the obstacle to the extended use of X rays, but now that they hoped to have, within the next twelve months, facilities at tuberculosis clinics both at the Adelaide and the Children's Hospital, by which practitioners could send doubtful cases for an X ray opinion and have the patient returned to them for treatment if necessary, it was opportune that they should hear Dr. Cowan's emphasis on the value of this method of investigation.

There remained the laboratory methods. But time permitted him only a brief reference to this aspect. In these early cases the sputum would often be "negative". They should not rule out the possibility of disease on that evidence alone, as Dr. Hone feared many of them did.

He had spoken of the Mantoux test, and of his sense of its value, especially in children. But, after all, both this and the single X ray film only assured them of infection in the past, but not necessarily of present active disease.

For these early cases the important treatment, of course, was rest, mental as well as physical. They faced the contradiction that the least movable parts of the lungs were the most likely to show evidence of active disease, yet the more they were rested the more likely they were to recover.

DR. J. WALTER BROWNE agreed with Dr. Cowan in the necessity for an improvement in their methods of getting patients with pulmonary tuberculosis under treatment at an early stage in their disease. Like every other medical man engaged in tuberculous work he had very frequently to lament the late stage in which he had found the patient on first examination. There were many reasons for this unfortunate state of affairs. Sometimes it was the fault of the patient. The illness was supposed to be trivial, and was treated as such for some time; or when it was recognized the patient was afraid of losing his job if he made known the fact that he had tuberculosis, or he kept at work too long for the sake of his dependants. Sometimes also, he regretted to say, the delay was due to wrong diagnosis, the illness being treated as something else—a nervous breakdown, or a simple pleurisy, or dyspepsia or anæmia, or debility.

The remedy for all this was improved education of both the public and of the profession, and Dr. Browne was of opinion that there had been a considerable change for the better in both respects within his recollection. He agreed with Dr. Cowan in the inestimable value of X ray examination at all stages of the disease, but he was not quite at one with him in deprecating the value of physical examination in the early stages as compared with the X ray findings. He was of opinion that there were changes in the percussion note and in localized movements of the chest wall in respiration at a very early stage indeed, perhaps quite as early as any indicated by the X ray film. Auscultatory changes probably occurred later.

He believed in the tuberculin tests, Mantoux or von Pirquet, as indicating infection, but seldom or never used the subcutaneous tuberculin test. He did not find it necessary in most cases, but was prepared to admit that in certain cases, such as those difficult ones so frequently found in returned soldiers, where there were many signs and symptoms which might be attributed to tuberculosis, but in which the sputum had never been found positive, this test might be found useful.

As regards treatment, Dr. Browne's practice was to regard every case with a positive sputum as not really early and to induce artificial pneumothorax at the earliest opportunity, if the case was otherwise suitable for such treatment.

Patients with a "negative" sputum might be treated on hygienic lines alone for, say, three months, and if obviously improving might be allowed to continue on such lines. If they failed to improve and were undeniably tuberculous then artificial pneumothorax should be induced. As regards sending early cases "to the north", Dr. Browne was not an advocate of such treatment. All patients in the early stages required emphatically to be kept under constant observation. The only patients, in his opinion, suitable for the north were the old cronies who had already undergone treatment and education in some suitable institution. They often seemed to do well.

DR. A. R. SOUTHWOOD complimented Dr. Cowan on the excellence of his paper. Dr. Cowan's work of many years had done much to educate practitioners on the subject of tuberculosis and especially on the need for early recognition and treatment of the disease. Progress in this matter depended so much on enlightenment of the whole community on the subject, and in individual cases the family medical attendant could do a great deal in instrucing the patient and his associates.

Dr. Southwood thanked Dr. Hone for his reference to the work of the boards of health. Most local boards took their responsibilities seriously, and did what they could. It was only through the cooperation of patients, private practitioners and health authorities that the best results could be obtained. Dr. Cowan's paper, and the discussion on it, would infuse greater enthusiasm into all concerned.

In reply, Dr. Cowan thanked Dr. F. S. Hone and the other speakers for their kindness and forbearance. The hour was late and he would reply quite briefly. He did not wish to convey the impression that he thought the health authorities generally were lax in carrying out their duties; but they were so in some instances. It was a rather one-sided arrangement. If a practitioner were caught in failing to notify a case, he received a very curt reminder and possibly was fined. The forbidding document he received demanding an explanation did not tend to promote the friendly relations that should exist between health authority and practitioner. He was inclined to agree with Dr. Ray Hone that compulsory notification had been a failure, but he would not suggest its abolition at the present stage. He was in agreement with Dr. Browne, who advocated earlier induction of artificial pneumothorax. This was the modern tendency, and he felt sure it was a wise one. He had not recommended that physical examination be neglected; but he did wish to emphasize that considerable pathological change might be present in the lungs with no demonstrable abnormal physical signs. This was the practitioners' most serious pitfall in diagnosis and could be avoided only by its frank recognition. His sympathies were with the practitioner. Even with every facility for diagnosis available, sometimes it was impossible to say for certain whether the symptoms complained of were due to tubercle bacillus infection or to some other cause. All he would ask of the practitioner was to be on his guard in cases of vague ill health in child or young adult, especially in those who had been in contact with an open case of pulmonary tuberculosis, and not to let up merely because there were no abnormal physical signs in the chest.

Pathological Specimens.

PROFESSOR J. B. CLELAND showed a series of pathological specimens in part illustrating various aspects of Dr. Cowan's paper on pulmonary tuberculosis. These comprised the following:

1. The lungs in extensive pulmonary tuberculosis with cavitation, in a girl of sixteen, who also had tuberculous mesenteric glands, tuberculous "dysentery" and tuberculous laryngitis.

2. An old tuberculous cavity in the lung with recent miliary spread following lobar pneumonia, in a man aged twenty-five, who had been quite well until fourteen days before admission to hospital, and who died from tuberculous meningitis five days later.

3. An organizing pneumonic exudate with which were associated caseous tuberculous foci. The patient was a male, aged twenty-five, who had had a cough for three months and who died from tuberculous meningitis.

4. Contracted amyloid kidneys and amyloid liver, from a male aged forty-two, who died from extensive pulmonary tuberculosis with cavitation.

5. Tuberculous laryngitis in a male, aged forty-one, with extensive pulmonary tuberculosis, with a history of cough for sixteen years and of hoarseness for two months.

Moderate Silicosis of the Lungs in a Miner.

The patient was a male, aged fifty-one, who had been a miner until two and a half years before death. He developed breathlessness, pain in the left side, and cough. His lungs showed extensive fibrosis with nodules of stony hardness, the right lung weighing 806·5 grammes (twenty-eight and a half ounces) and the left 778·2 grammes (twenty-seven and a half ounces). He died from failure of the right side of the heart.

Advanced Silicosis of the Upper Lobes of Both Lungs Suggesting a Mediastinal Tumour.

The patient, a male, aged about forty years, under the care of Dr. E. West, had frequent vomiting and cough. He had been a miner. X ray photographs showed a dark shadow apparently bulging from each side of the superior mediastinum and suggesting a neoplasm. *Post mortem* examination showed, however, extensive silicosis in the upper lobes of the two lungs, which had led to much reduction in the size of these lobes and their contraction towards the lung roots. Their centres were occupied by dense, very hard, grey masses of fibrous tissue. The upper portions of the lower lobes of the lungs on each side had undergone compensatory emphysema, and at death occupied the apical portion of each pleural cavity instead of the upper lobes doing so.

Primary Carcinoma of the Lung. Death from Deposits in the Brain.

The patient, E.S., aged forty-six, a nurse, was admitted to hospital under Dr. Cowan's care on November 25 with a history that she had developed pain in the left side of the chest six weeks previously; at that time it was thought to be due to fibrositis. The pain became worse, was aggravated by deep breathing, she developed a slight cough and a little sputum was expectorated. Three weeks before admission three pints of fluid were aspirated from the chest, and she had been easier since then until two days before admission, when the pain in the chest became more severe, followed in a few hours by a slight hæmoptysis and that evening by a fairly severe hæmoptysis, accompanied by a slight sweat. Her weight had gone down during the six weeks.

On examination her temperature was 38·2° C. (100·8° F.), her pulse rate 112 and her respiratory rate 24. There were signs of extensive consolidation of the upper part of the left lung with fluid below the fourth intercostal space and no other abnormality. X ray examination showed a fairly large encysted effusion in the left side of the thorax, occupying the base and outer portion of the lung field, and displacing the heat towards the right side. Twenty-three ounces of fluid were aspirated from the left thorax; examination of the fluid showed a little macroscopic blood with, microscopically, a definite increase in lymphocytes, some polymorphonuclear cells, red blood cells and endothelial cells. No microorganisms were detected either on direct smears or cultures. The sputum examination revealed no tubercle bacilli. The condition was regarded as one of tuberculous pleurisy and pulmonary tuberculosis, and pneumothorax was done on December 6, the subsequent X ray examination revealing some adhesions. The chest was refilled with air on three occasions, the last being on January 9. The intradermal tuberculin test had been done on November 28 and yielded no reaction. Throughout the course she had a hectic pyrexial temperature and gradually went down hill. On January 10 she could not move her right hand; this was thought to be hysterical. Two days later the paralysis spread to the wrist, and after another two days to the elbow, and on February 1 she was incontinent, with general paresis of an upper motor neurone type of the right side. A week later her condition was somewhat worse, but examination of the fundi showed no abnormality. She died on February 11, just about fourteen weeks from the onset.

At the autopsy a primary carcinoma of the lower lobe of the left lung was found. Extension had occurred to the left pleura, which was studded with nodules. A few miliary specks of malignant deposits were present in the right lung. There were secondary deposits up to 2·5 centimetres (one inch) in diameter in the liver, and small ones in the suprarenal glands and pancreas. The brain showed several hæmorrhagic deposits. The first and largest was 2·5 centimetres in length by about 1·8 centimetres in width by 2·5 centimetres antero-posteriorly. This oblong mass of growth came to the surface of the left cerebral hemisphere in the centre of the pre-Rolandic gyrus, 4·3 centimetres down from the superomedial border of the hemisphere. It extended forward almost to the precentral sulcus and back into the postcentral gyrus. The mass ran downwards and inwards to within six millimetres of the lateral part of the *corpus callosum* and 1·25 centimetres above the caudate nucleus and the internal capsule. Another degenerated cystic hæmorrhagic deposit was present at the anterior end of the right frontal lobe, extending to within 1·25 centimetres of the frontal pole and being about 1·8 centimetres in diameter, and the last deposit was a fleshy-looking mass, 1·8 by 2·5 centimetres, in the antero-lateral extremity of the right cerebellar lobe, invading the *arbor vitæ* and destroying the structure of the peduncles on that side and interfering with the nutrition of the dentate nucleus, portion of which, with its surrounding structures, was occupied by a necrotic mass fully 2·5 centimetres in diameter.

Microscopically the growth proved to be a carcinoma, the cells tending to be rather spindle-shaped to spherical or short columnar, and being arranged in a vague alveolar fashion. Sections from two places in the cerebrum showed merely hæmorrhage, but the deposit in the cerebellum showed large epithelial cells, some almost columnar, occasionally arranged round a definite lumen.

A Fibromyoma Separate from the Uterus and on the Lateral Wall of the Pelvis.

The specimen was from a woman, aged forty-six years, who died from a primary carcinoma of the lung with deposits in the brain. On the lateral wall of the pelvis on the left side was a fibromyoma measuring 6·25 by 3·1 by 3·1 centimetres (two and a half by one and a quarter by one and a quarter inches). This was quite separate from the uterus, though it probably had originated as a subserous pedunculated tumour of that organ. It had undergone considerable degeneration in parts.

Fracture of the Femur.

DR. L. O. BETTS demonstrated a case of a fractured neck of the femur in a man of thirty-five years. He had a basal impacted fracture, with the neck almost at right angles to the shaft. This had remained impacted for seven weeks, and had distinct callus formation. The fracture had been broken down, heavy traction applied in the abducted position to overcome the half inch of shortening, and the limb put up in a Whitman plaster. X ray examination showed that reduction was absolute, and at twelve weeks after the injury the man was getting about in a weight-bearing caliper. The case was shown to illustrate the exact reduction that could be obtained even in late cases by the Whitman method.

Obituary.

SAMUEL WILLIAM HENRY SUMMONS.

DR. SAMUEL WILLIAM HENRY SUMMONS, whose death, as recorded in a previous issue of this journal, occurred on April 30, 1933, was born at Carlton, Victoria, on June 22, 1878. He was the son of the late Samuel Summons,

Senior Inspector of Schools, of Victoria. Shortly after Summons was born his parents moved to Ballarat, where his education commenced. He was brought back to Melbourne at the age and eleven and was sent to Kew High School. Later on he was sent to Scotch College. From here he matriculated and went as an undergraduate to the University of Melbourne to study medicine in 1897. During his course he secured various honours and won the Beaney Scholarship in Pathology at the final honours examination in 1902.

After graduation Summons became Resident Medical Officer and later Medical Superintendent of the Children's Hospital, Carlton. He held this position for four years. He was assistant to Sir Richard Stawell for two years and acted for a space as Medical Tutor at Trinity College within the University of Melbourne. Prior to starting practice, he travelled to England for post-graduate study. After his return he began practice at Kew in 1909 and continued in active general practice to within three days of his death.

For some years Summons held an honorary position on the staffs of the Children's Hospital, Carlton, and Saint Vincent's Hospital, Melbourne; his failing health eventually compelled him to resign these positions. He was whole-hearted in his service to his patients and there is no doubt that his end was hastened by his frail constitution not being able to stand the strain of all that he felt it his duty to attempt. During the war he was not able to leave Australia for service overseas, but did a great deal of home service. He carried out much onerous and responsible work in connexion with the meningitis epidemic. During his later years his chief recreation was found in his garden at Kew and at his seaside home at Flinders.

Summons belonged to a gifted family. He had the joy of practising close to his two brothers; they did much professional work together. He will be missed by a large circle of friends and by his many patients. The sympathy of the medical profession is extended to his widow and family and to his two brothers.

Dr. Konrad Hiller writes:

My first association with Harry Summons was when we were students together over thirty years ago. The friendship then made persisted till the time of his death. After graduation and periods of residentships he was appointed to the honorary staff of the Children's Hospital and also occupied a position as out-patient physician to Saint Vincent's Hospital. To these institutions he gave of his best and laboured hard for a number of years in their interests. Never robust, however, the strain of hospital work, together with a steadily increasing private practice, began to tell on his health, and with great reluctance he ultimately decided to withdraw from his hospital positions and devote himself to his practice. Though relieved by this action, it was a blow to his aspirations, but, having made the sacrifice, he accepted the more limited sphere with fortitude. Henceforth he devoted his strength to his practice and his home. Well equipped by his wide hospital experience, conscientious in all his work, he built up a sound practice, in which his patients loved and trusted him. Of a retiring disposition, he never paraded his feelings, but when occasions arose he expressed his opinions and held to them with a tenacity born of conviction. Precluded from strenuous exercise by ill health, he was still able to do gardening, of which he was fond, and spent many days in this pursuit at his seaside house at Flinders. Cut off at a comparatively early age, he leaves a widow and family, to whom sincere sympathy is extended by all his friends.

Dr. A. E. Rowden White writes:

After a brilliant course at the University of Melbourne, Harry Summons, as everybody knew him, was appointed to the Children's Hospital as a junior medical officer. In the course of time he became senior resident medical officer, a position akin nowadays to that of medical superintendent. His organising ability, and his good work in that capacity, earned for him, on entering private practice, a position on the honorary medical staff of the Children's Hospital, a post he held for many years.

About the same time he joined the staff of Saint Vincent's Hospital at its inception as a clinical school, and did excellent work in the clinical instruction of the students of the time. By his pleasant manner and lovable character he endeared himself to all who came in contact with him.

I have very happy recollections of him as a colleague in our resident medical officer days at the Children's, and later on for years when we were associated on the honorary medical staffs of the Children's and Saint Vincent's Hospitals.

Correspondence.

AN ECONOMIC COMMENTARY AND DIAGNOSIS.

SIR: To the reader who is familiar with economic literature and the opinions of eminent authorities who approach the subject with a due sense of its immensity and far-reaching relations, the lettter of "M.D., C.M." will be amazing reading. Indeed, the very assertive and over-confident manner in which he meets and disposes of problems that are exercising the acutest minds in the world today cannot, to say the least, be very convincing to your readers. Medical men belong to a class trained in the art of reasoning and relating cause to effect with some precision, and must view with suspicion the expoundings of one who can at best be described as "an interested outsider" on subjects outside his professional ambit. The economic problems of the day must possess great interest for all of us, and we even have a right to our own opinions and views, but to speak of Money Moloch's lions, as represented by the trusted commissions of inquiry, deliberately stifling the truth in their findings, seems fantastic and unutterably absurd.

Let us select some further passages: "The application of science to industry has changed the function of money, which formerly was the measure of exchange of goods and services." At what point did the change-over take place? Are not goods and services still exchanged in this manner? Do principles which are plain enough in the simpler relations between men, not hold good in the more complex processes of commerce? If not, when precisely do they fail? As for the A + B theorem, the knock-out blow, as it were, to all orthodox reasoning, it certainly is difficult to follow the writer's meaning, "purchasing power is distributed in wages, salaries and dividends only". Why the payments for raw material, the bank charges (paid out later in dividends to shareholders of banks), rent to landlords *et cetera* should not be regarded as purchasing power is difficult to understand. Nevertheless, this amazing theorem is held to be self-evident and above argument, out of bounds, so to speak. The statement that "unemployment can never again be cured by employment" is too paradoxical, or is it a riddle?

It will appear to many readers that "M.D., C.M." is dealing with a maldistribution rather than a deficiency of purchasing power. That it is plentiful, though not fairly spread, may be seen if and when, for instance, an approved issue of first class securities is listed on the exchanges. Some part of this may even be traced back to the part B of Douglas's outgoings which he calls not purchasing power, although it is difficult to detect the difference. Taken as a whole, the letter gives the impression that the disciples of Douglas are mistaking shadow for substance. As a lubricant is necessary for the smooth running of a machine, so money is helpful in the process of business. In either case, other than a lubricant, it is powerless for good, and if applied in reckless profusion may work incalculable injury, as some European countries know to their cost. It is significant that in the recent election in Germany every one of the parties appealing for votes gave an unqualified pledge that it would stand at any cost against any increase of paper money in circulation. They knew.

Finally, I trust that "M.D., C.M." will believe that I am as conscious as he of gross inequalities in the life of the people, and quite as earnest in hoping for their removal.

As he has put questions to Dr. Dane, I may be permitted to ask him two.

Where may be seen the accumulated products of past years that the theorem declares cannot have been sold?

Why am I, as an owner of bank shares, receiving a bare 3½% on my invested capital?

Yours, etc.,

J. A. BISSET.

16, Collins Street,
Melbourne,
June 3, 1933.

Corrigendum.

In the paper by Dr. Vera Krieger on the effect of molybdenum impurity in the arsenophosphotungstic acid reagent on the uric acid content of blood, published in the issue of June 3, 1933, an error appears in the box headings of Table VI. The third box heading should read: "Uric Acid from Thioneine + Indirect"; the sixth box heading should read "Direct – Indirect Method".

Books Received.

A SHORT HISTORY OF DENTISTRY, by L. Lindsay; 1933. London: John Bale, Sons and Danielsson, Limited. Crown 8vo., pp. 88. Price: 3s. 6d. net.

A SYNOPSIS OF SURGERY, by E. W. Hey Groves; Tenth Edition; 1933. Bristol: John Wright and Sons, Limited. Crown 8vo., pp. 701, with illustrations. Price: 17s. 6d. net.

STREPTOCOCCI IN RELATION TO MAN IN HEALTH AND DISEASE, by A. W. Williams, M.D.; 1932. London: Baillière, Tindall and Cox. Royal 8vo., pp. 272, with 8 plates. Price: 29s. net.

Diary for the Month.

JUNE 27.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JUNE 28.—Victorian Branch, B.M.A.: Council.
JUNE 29.—South Australian Branch, B.M.A.: Branch.
JUNE 29.—New South Wales Branch, B.M.A.: Branch.
JULY 3.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JULY 4.—New South Wales Branch, B.M.A.: Council, quarterly.
JULY 5.—Western Australian Branch, B.M.A.: Council.
JULY 5.—Victorian Branch, B.M.A.: Branch.
JULY 6.—South Australian Branch, B.M.A.: Council.
JULY 7.—Queensland Branch, B.M.A.: Branch.
JULY 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JULY 14.—Queensland Branch, B.M.A.: Council.
JULY 18.—New South Wales Branch, B.M.A.: Ethics Committee.
JULY 19.—Western Australian Branch, B.M.A.: Branch.
JULY 19.—Victorian Branch, B.M.A.: Clinical Meeting.
JULY 20.—New South Wales Branch, B.M.A.: Clinical Meeting.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xvi, xvii and xviii.

FREMANTLE HOSPITAL, FREMANTLE, WESTERN AUSTRALIA: Junior Resident Medical Officer.

LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officers.

MOSSMAN HOSPITAL, MOSSMAN, NORTH QUEENSLAND: Assistant Medical Officer.

NEWCASTLE HOSPITAL, NEWCASTLE, NEW SOUTH WALES: Medical Superintendent, Junior Resident Medical Officer.

NEW SOUTH WALES COMMUNITY HOSPITAL, SYDNEY, NEW SOUTH WALES: Medical Officers.

ROYAL PRINCE ALFRED HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Officers, Bacteriologist.

THE BRISBANE AND SOUTH COAST HOSPITALS BOARD, QUEENSLAND: Honorary Officers.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Combined Friendly Societies, Clarendon and Kangarilla districts. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor", THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad per annum payable in advance.